EVALUATION OF LEFT VENTRICULAR FUNCTION IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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MEDICAL COLLEGE, SALEM

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CERTIFICATE

This is to certify that this dissertation **"EVALUATION OF LEFT VENTRICULAR FUNCTION IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE"** is a work done by **Dr. R. PRAVEEN BABU** under my guidance during the period of 2010 - 2013. This has been submitted to the partial fulfilment of the award of M.D. Degree in General Medicine (Branch I) by the Tamil Nadu Dr. M.G.R Medical University, Chennai-32.

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DECLARATION

I solemnly declare that this dissertation **"EVALUATION OF LEFT VENTRICULAR FUNCTION IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE"** was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem - 636 030 under the guidance and supervision of **Prof. Dr. S. RAMASAMY,** M.D., Professor of General Medicine, Govt. Mohan Kumaramangalam Medical College and Hospital, Salem. This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in fulfilment of the University regulations for the award of the degree of M.D. General Medicine (Branch I)

Place : Salem

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CONTENTS

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) remains a major public health problem. It is projected to rank fifth in 2020 in burden of disease caused worldwide according to WHO study.

In recent years, importance in understanding and empirical management of COPD has increased manifold considering the gravity of the situation.

The concept of cor pulmonale i.e. involvement of right side heart usually occurs in COPD. Recently published studies claim that along with right side, left heart involvement too occurs resulting in complete heart failure.

Sufficient studies have not been conducted, or since many current studies are in process, it has not been proven yet. Percentage of documentation of left heart involvement in COPD is not upto the mark in India. Thus, the aim of our research is to ascertain the recent findings by undertaking a case study of our own among COPD patients.

AIM AND OBJECTIVES

To evaluate the left ventricular systolic and diastolic functions of Chronic Obstructive Pulmonary Disease patients using Transthoracic Echocardiography.

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE

The beginning of modern chest medicine can be traced to the classic volume by Laennec, "A treatise on diseases of the chest" which appeared in 1821, laid the corner stone of modern chest medicine.

In his treatise, Laennec, devoted a chapter to "Pulmonary Catarrh or Bronchitis" and emphysema. The chapter on bronchitis distinguishes between acute and chronic form and sub divides chronic bronchitis into two types – the humid (Copious Expectoration) and dry (Scarcely any Expectoration). He identified "Dilatation of air cells" as the essential feature of emphysema.

Recognition of chronic bronchitis as a potentially grave illness rather than as a trivial one, had to wait the "London Fog" of 1953, which was brought about by bad weather and air pollutants, carried with it a surge in morbidity and mortality due to chronic lung disease.

After World War II, clinical investigations of pulmonary disease were provided with a new diagnostic armamentarium. Pulmonary Function tests were extended beyond simple Spirometry and innovative

techniques were developed for assessing the distribution of gases within the lungs which greatly improved our understanding of COPD.

COPD Definition :

The GOLD guidelines define COPD as "A preventable and treatable disease with some significant extra pulmonary effects that may contribute to the severity in the individual patient. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases"[1].

Epidemiology :

Globally, COPD is one of the major cause of morbidity and mortality. According to 'The Global Burden of Disease Study' COPD is expected to become the 3rd most leading cause of death and the 5th leading cause of loss of 'Disability Adjusted Life Years' (DALYs) in the recent future.[2]

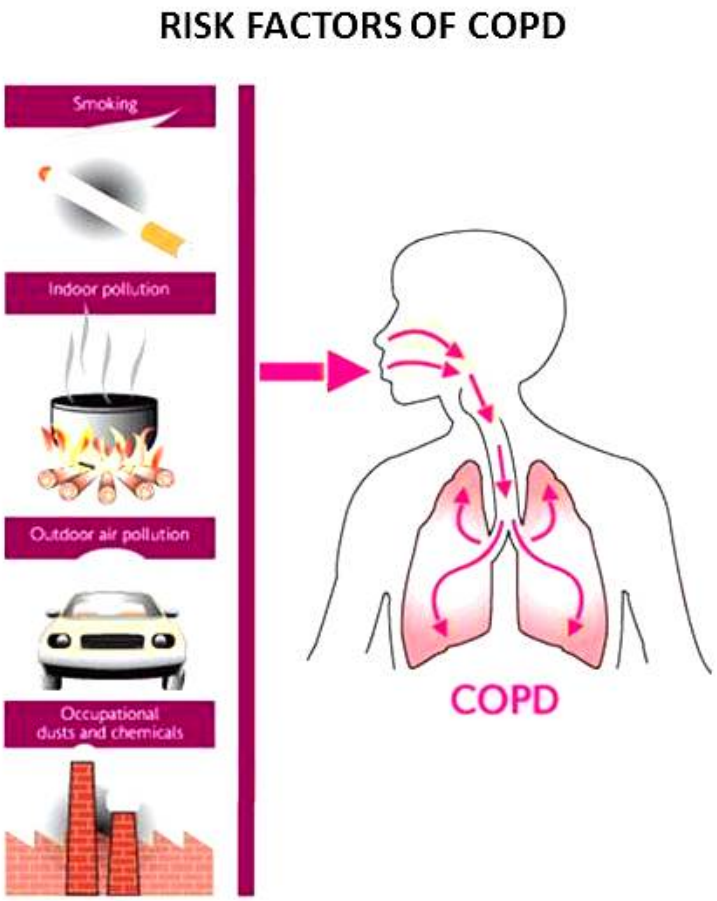
WHO data on prevalence of COPD is 3.8% in Asian countries. But to the contrary, according to recent reports, the prevalence of COPD is 6.3% in 12-Asian Pacific countries[3].

The Indian Council of Medical Research (ICMR) took the initiative to study the epidemiology of chronic respiratory diseases and sponsored the Indian study on Epidemiology of Asthma, Respiratory symptoms and Chronic bronchitis (INSEARCH) which included 4-centres in the Phase I and 12 other centres in the Phase II study.

The results of the Phase I study from Chandigarh, Delhi, Kanpur and Bangalore reported the overall prevalence rates of 5.0 and 3.2 percent in men and women respectively, of and over 35 years of age.[4]

RISK FACTORS:

- Smoking
- Indoor air pollution
- Occupational dusts
- Infection
- Ageing
- Genes



Smoking has traditionally been known to be the most important cause for COPD amounting to almost 85% of the COPD cases (50% smokers develop COPD)[7,8,9], the rest being classified as non-smoking COPD (15%)[5,6].

In developing countries such as India, COPD due to non-smoking causes account to 30-50% of all COPD cases[10].

Burning biomass fuel such as wood, cow-dung and crop-residues leads to release of air pollutants like SO_2, CO, NO_2, formaldehyde and

particulate matter smaller than 10 micron in size (PM10) in the ambient indoor air[10,11]. Chronic exposure to these pollutants has been shown to lead to COPD.

Another important risk factor for non-smoking COPD is prolonged exposure to occupational smoke/dust.

Tuberculosis is increasingly getting recognized as a risk factor for COPD[5,6,10]. It has been found that even after adequate anti-Koch's therapy in tuberculosis patients, they are 2-6 times more prone to develop airway obstruction. Spirometry and insidious onset of symptoms similar to COPD in T.B. patients suggest the same.

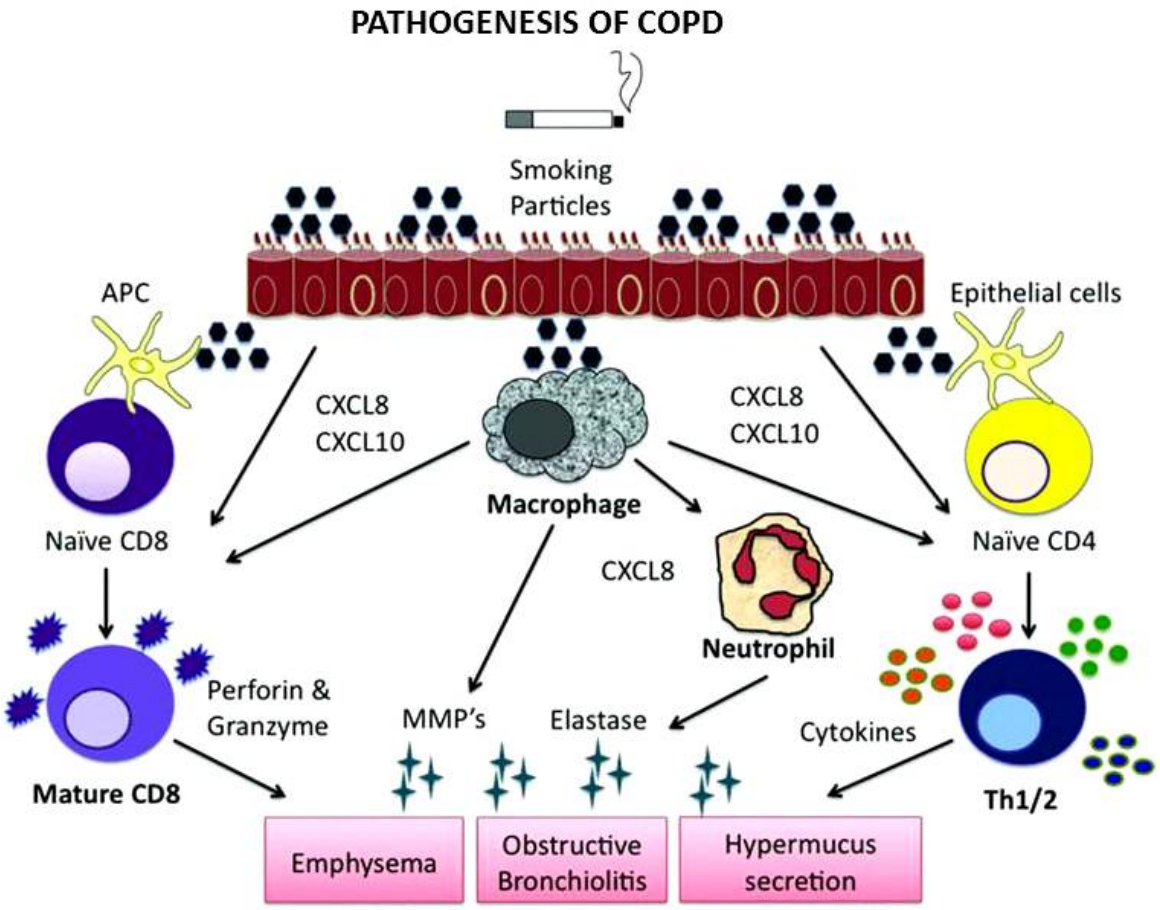
Recurrent respiratory infections in childhood have also been shown to be associated with development of COPD in adult age[5,6,10].

Physiological lung function decline induced by ageing can also predispose to COPD[5].

Genetics come to answer the question why only a fraction of smokers develop COPD, while others do not. Perturbation in SERPINA1 gene leads to deficiency of AAT-1, causing uninhibited action of proteases culminating the development of emphysema[12].

Pathogenesis of COPD

The cells primarily involved in COPD inflammation are neutrophils, macrophages and lymphocytes. The inflammatory cells release a battery of inflammatory mediators like cytokines, chemokines and chemoattractants which perpetuate the inflammation leading to an uncontrollable cascade.



Neutrophils by releasing chemoattractants like interleukin-8 (IL-8) and leukotriene B4 (LTB4) attract more neutrophils to the site.

Proteolytic enzymes such as elastase, proteinase-3, cathepsin G, cathepsin B and matrix metalloproteinases (MMP) released by neutrophils cause damage to elastic lung tissue[13].

Macrophages release[14]

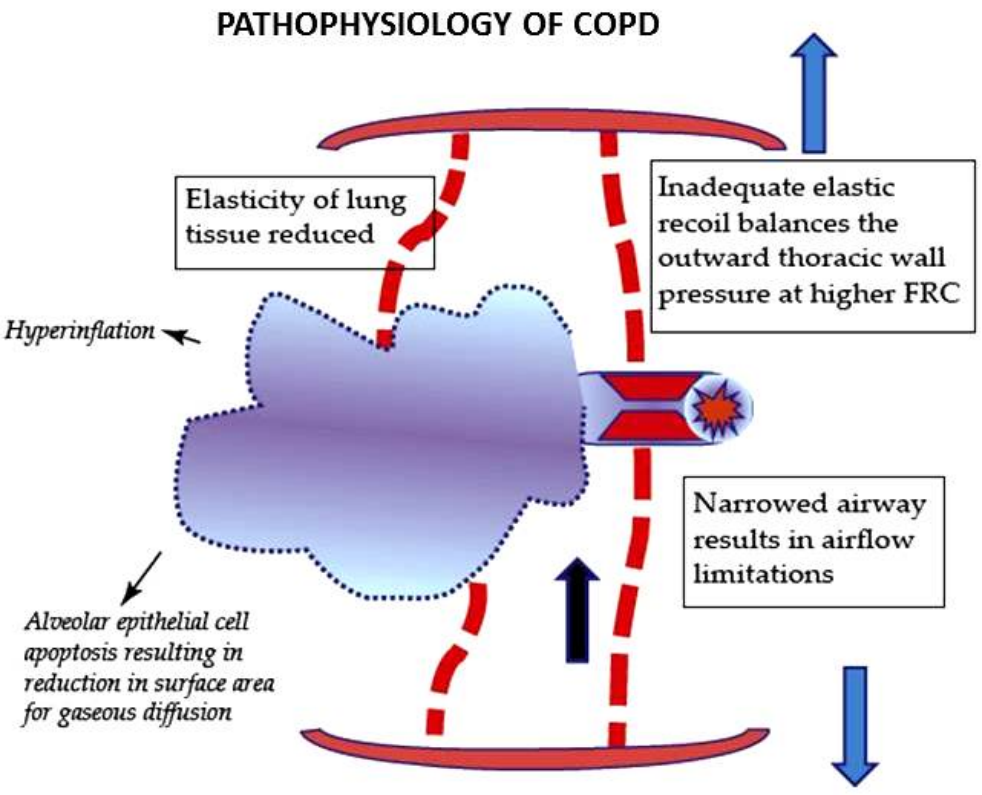
- cytokines and chemokines such as IL-8, IL-6, IL10, TNFα, LTB4 etc which attract and activate various inflammatory cells
- series of proteinases, particularly MMPs such as MMP-2, MMP-9, MMP-12, MMP-14, with tremendous elastolytic potential
- elastinolytic cysteine proteinases such as cathepsin K, L and S.
- reactive oxygen species

CD8+ lymphocytes release destructive enzymes such as perforin and granzyme B which has the ability to induce apoptosis of the alveolar epithelial cells and CD4+ lymphocytes induce autoimmune response toward the lung tissue[15].

Pathophysiology in COPD[16,17,18]

Elastin proteolysis

Elastin proteolysis results in reduction of elastic recoil pressures in the lungs moreover since the integrity and movement of air in the bronchioles are primarily reliant on elastic recoil pressures induced by surrounding elastic tissue, the damage to elastin in COPD results in significant airway narrowing with reduction in air flow to the bronchioles leading to air trapping in lungs.



Fibrotic remodelling

Fibrotic remodelling of the airways results in fixed airway narrowing causing increased airway resistance which does not fully revert even with bronchodilators.

Pulmonary Apoptosis

Extensive alveolar and bronchiolar epithelial cell damage and pulmonary capillary apoptosis are the histological features in emphysema and the physiological feature is decreased surface area of alveoli for gaseous exchange and ventilation - perfusion mismatch (V/Q).

Clinical Features

1. Chronic cough, continuous and productive, increased during early morning, may be intermittent and non-productive
2. Breathlessness on exertion, initially intermittent but later becomes persistent
3. Sputum production of any pattern
4. Frequent exacerbations of bronchitis

5. A history of exposure to risk factors, especially tobacco smoke, occupational dusts, home cooking and biomass fuels.

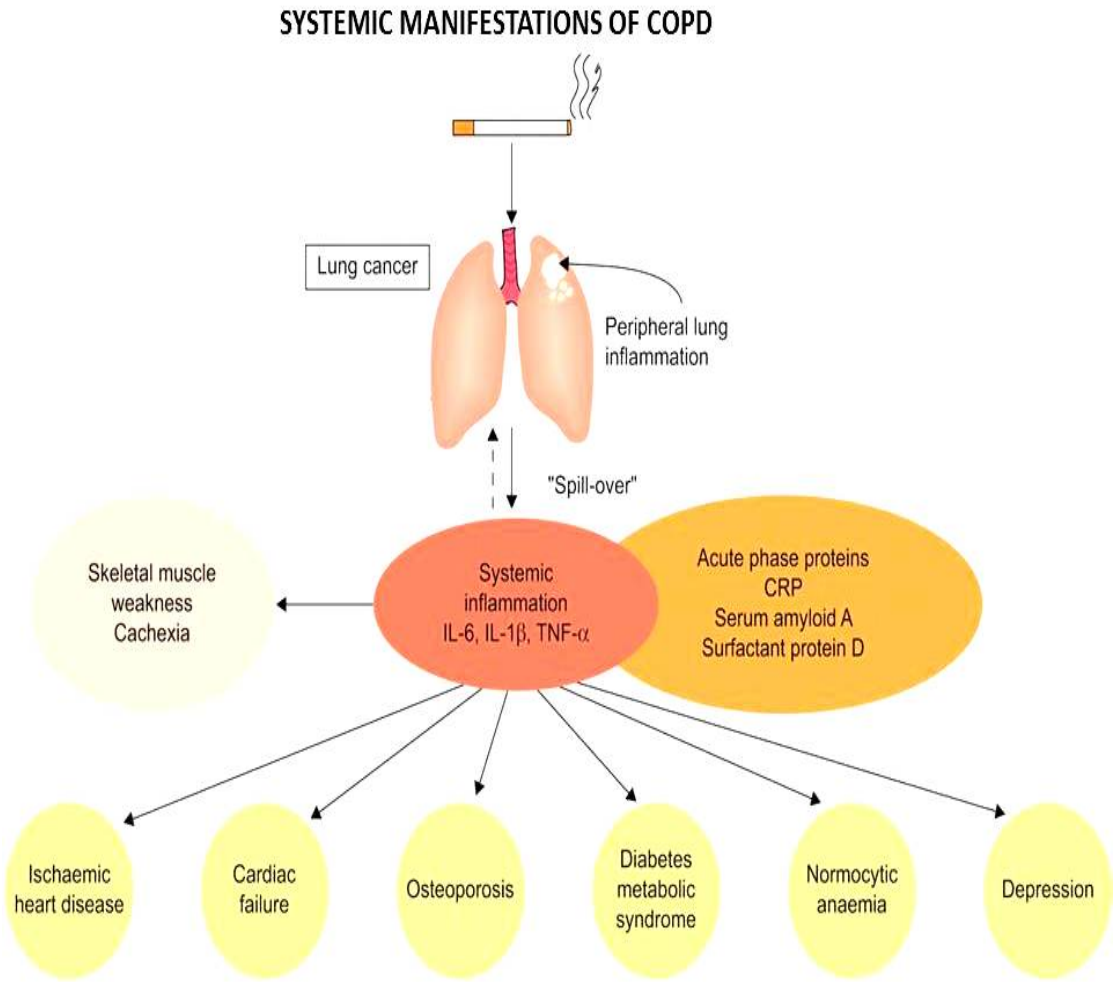
Systemic Manifestations of COPD[19,20,21]

COPD is never a disease of lungs alone. Mortality and morbidity in COPD patients is mainly due to the associated comorbities that occurs hand in hand with lung dysfunction. The common comorbidities associated with COPD are as follows:

1. Skeletal muscle wasting
2. Cachexia
3. Lung cancer (small cell, non-small cell)
4. Pulmonary hypertension
5. Ischaemic heart disease: endothelial dysfunction
6. Congestive cardiac failure
7. Osteoporosis
8. Normocytic anaemia
9. Diabetes mellitus/ Metabolic syndrome

10. Obstructive sleep apnoea

11. Depression



The markers of systemic inflammation that are increased in COPD patients are hs C-Reactive Protein (hsCRP), fibrinogen, ferritin, total leucocyte count, Reactive Oxygen Species, Interleukins, Transforming Growth Factor β1 (TGF-β 1) and Tumor Necrosis Factor-α (TNF-α) and other cytokines.

COPD and Heart

The comorbidities occuring in heart of COPD patients are of two types. First, diseases that occur because of sharing similar risk factors like smoking i.e. Coronary artery disease and Congestive heart failure. Other type is the diseases that occur secondary to COPD itself i.e. Secondary pulmonary hypertension and Ventricular Dysfunction.

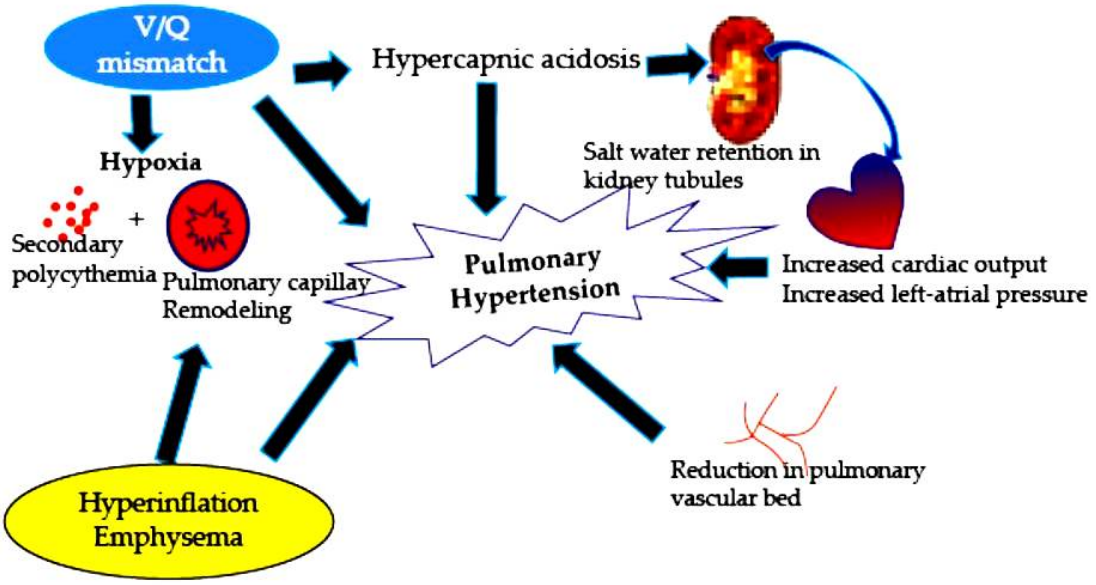
Traditional teaching is that mortality in early stages of COPD is due to cardiovascular disease whereas in late stages, it is due to respiratory problems. Coronary artery disease is common in COPD patients due to persistent low grade inflammation leading to atherosclerosis.[22]

Pulmonary arterial hypertension (PAH) in COPD occurs due to many reasons[23].

1. Hyperinflation and Alveolar hypoxia
2. Increased intra thoracic pressure
3. LV diastolic dysfunction

Alveolar hypoxia induces pulmonary artery vasoconstriction resulting in vascular remodelling which increases pulmonary vascular resistance.

MECHANISM OF PULMONARY HYPERTENSION IN COPD



Rise of intra thoracic pressure due to hyperinflation exceeds the venous pressure leading to reduction in blood volume to right and left ventricle resulting in increased pulmonary artery wedge pressure.

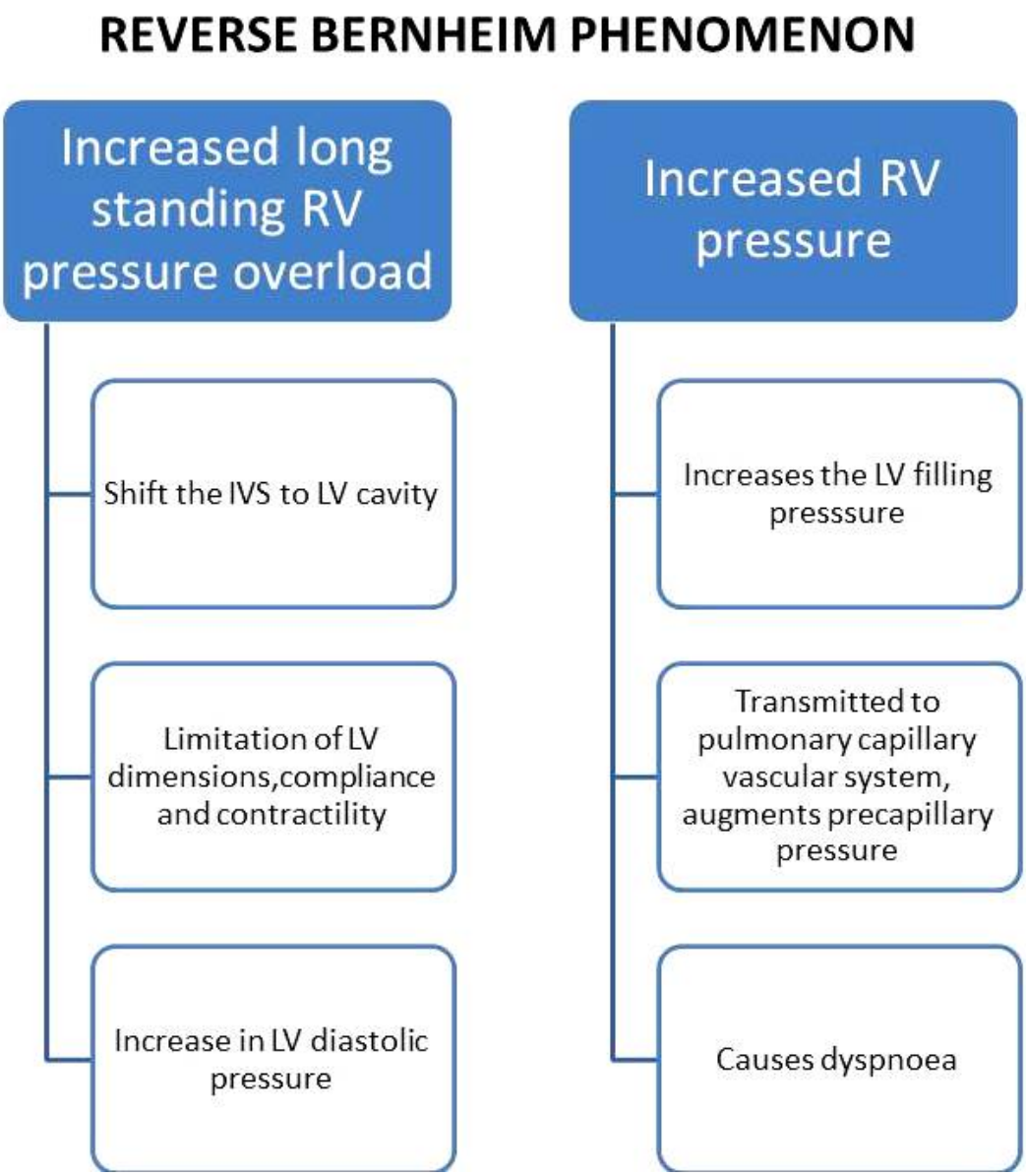
LV diastolic dysfunction causes back pressure on the pulmonary vasculature and increases pulmonary vascular resistance.

Chronic pulmonary arterial hypertension increases right ventricular after load and results in a clinical syndrome of right heart failure or corpulmonale.

Development of right ventricular hypertrophy and eventual right side heart failure is common in COPD. However disturbance in left ventricular function has been observed by several workers amongst

COPD patients. The left ventricular dysfunction seems to be an aftermath of right heart involvement.

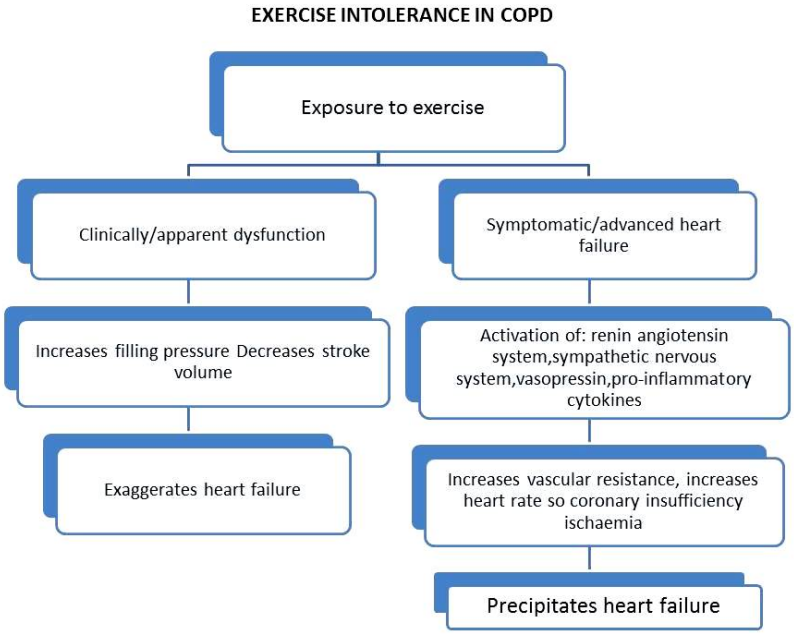
The influence of right ventricular volume/pressure overload on left ventricular function is explained by Reverse Bernheim phenomenon[24].



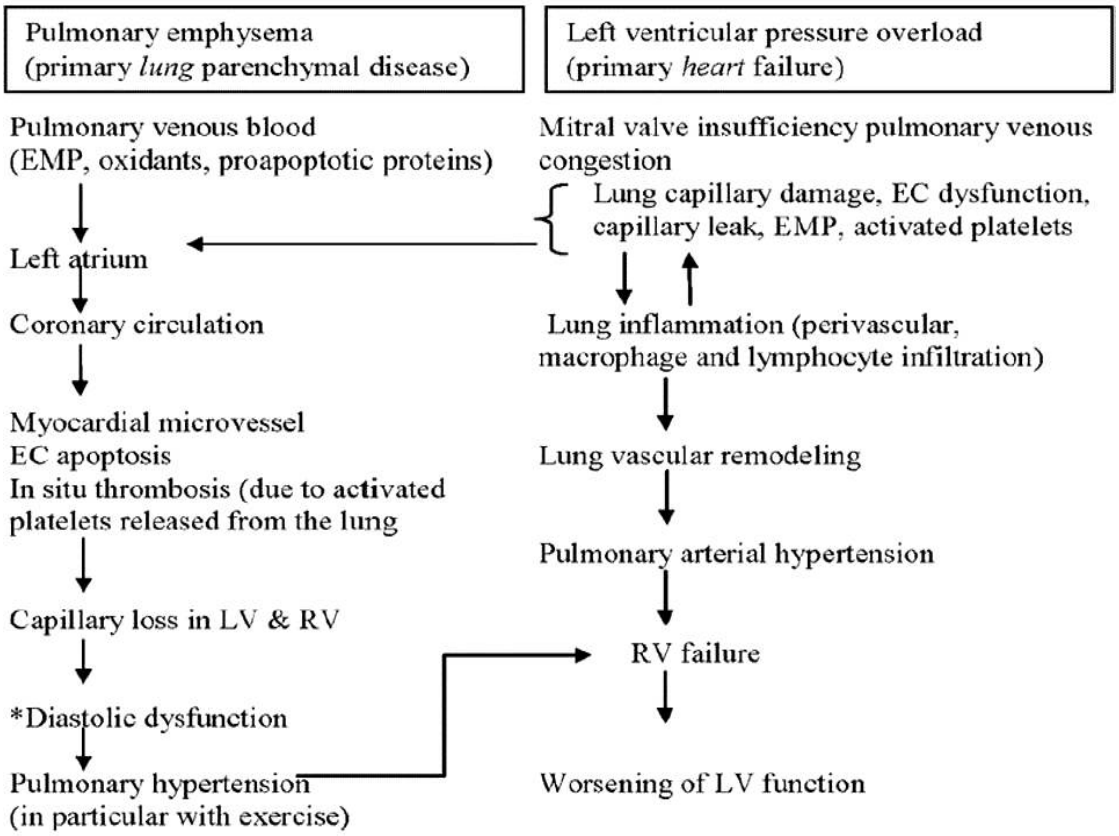
The predictors of diastolic dysfunction are:

1. Disproportionate tachycardia
2. Easy fatigability
3. Tachypnea
4. Shortness of breath

Though patients are asymptomatic even with impaired left ventricular function it is exposed when they exercise. Increased demand of tissues during exercise elevates the sympathetic drive. The extra pressure on the functioning of the heart is not met out due to adverse lung function[25,26].



MECHANISM OF DEVELOPMENT OF PHT IN PRIMARY LUNG PATHOLOGY(LEFT) AND HEART DISEASES(RIGHT)



EMP - Endothelial Micro Particles EC - Endothelial Cell

INVESTIGATIONS:

CHEST X RAY:

Chest X ray shows cardiomegaly in LVF patients with cardio thoracic ratio greater than 0.5 but reliability of X ray in COPD patients is poor due to hyper inflation.[27]

Usually COPD patients have tubular heart. Non tubular heart in X-ray of COPD patients should raise the suspicion of diastolic

dysfunction. This finding has no relation with FEV_1 or increased broncho vascular markings. FEV_1

CXR OF COPD PATIENT SHOWING TUBLAR HEART

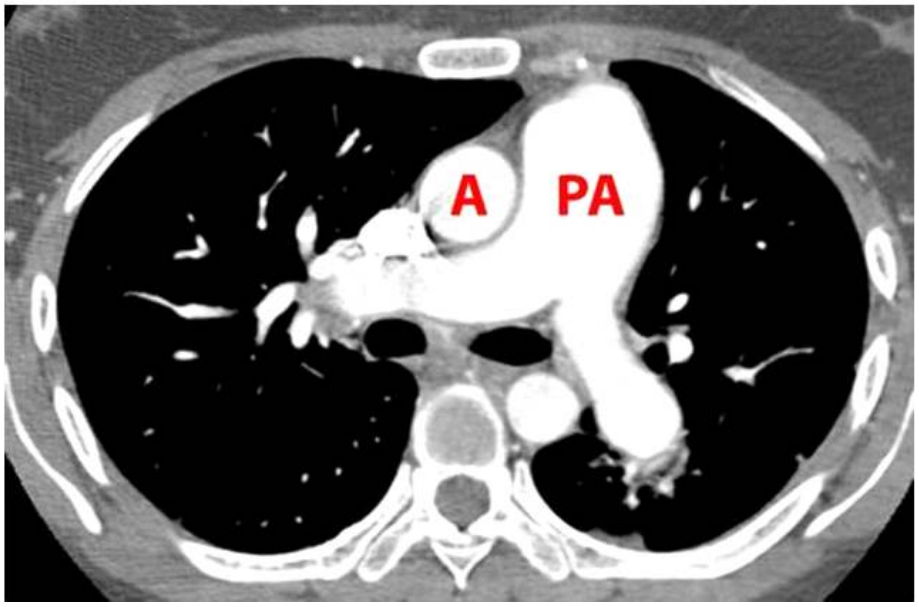
COPD PATIENT HAVING NON TUBULAR HEART

In chest X ray lateral view obliteration of retro sternal space implies right ventricular hypertrophy.

CT SCAN:

In mediastinal window, if the pulmonary artery diameter is same as the aorta suspect pulmonary artery hypertension. The specificity of this finding in CT is 100%.

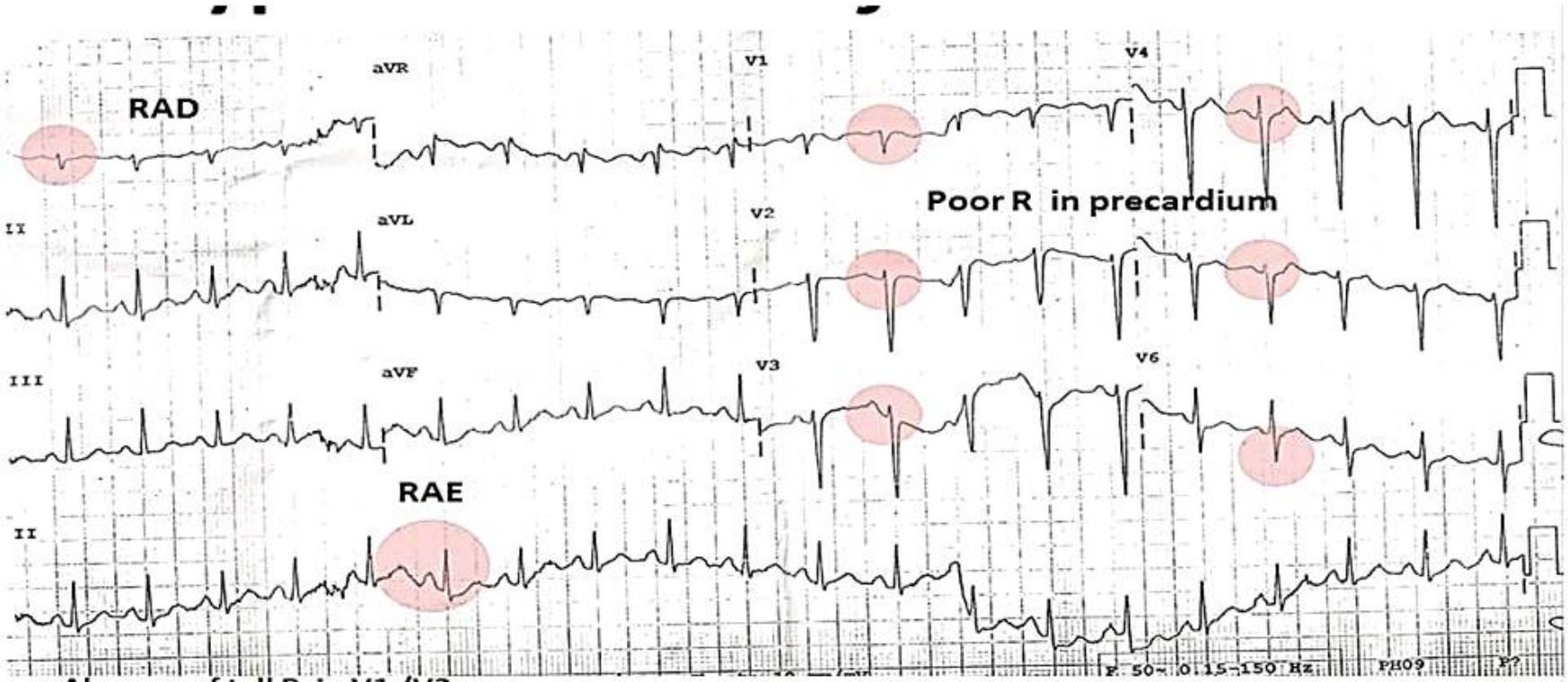
CT CHEST SHOWING PULMONARY ARTERIAL HYPERTENSION



ECG:

ECG shows right axis QRS deviation, P pulmonale and right ventricular hypertrophy.

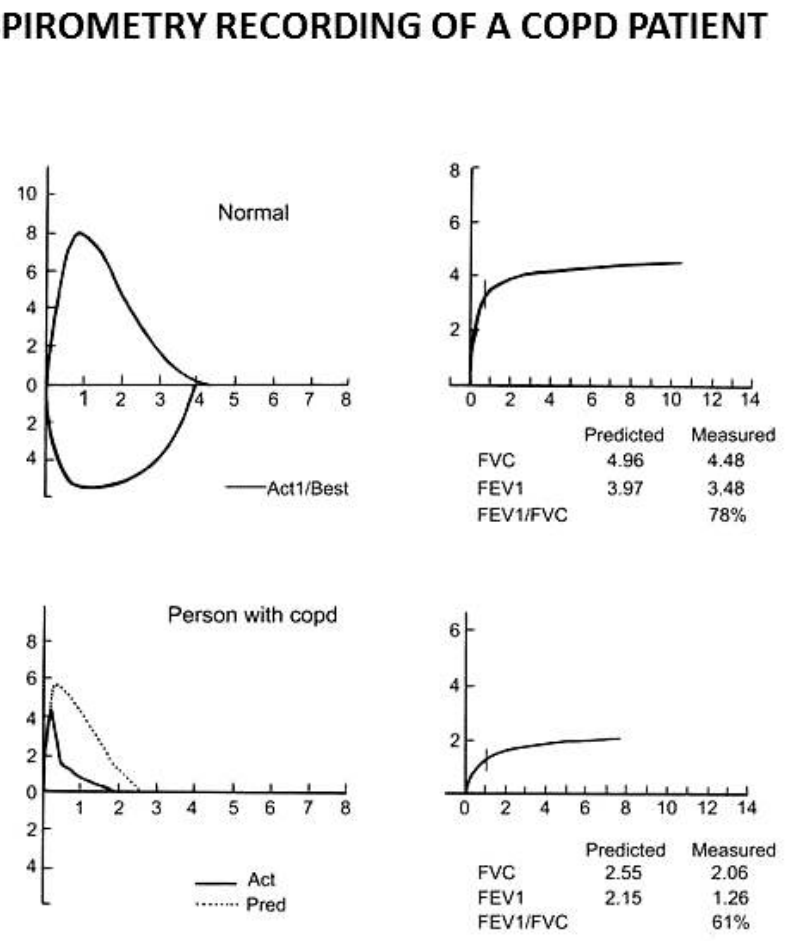
ECG OF COPD PATIENT SHOWING TYPE C RIGHT VENTRICULAR HYPERTROPHY



- Absence of tall R in V1 /V2.
- Clockwise rotation with transition zone pushed to extreme left
- Mimics anterior MI
- Still , RAE and right axis deviation help us to diagnose RVH .
- S in lead 1 , 2, 3, if present is classical of Type C RVH

PFT:

PFT shows decrease in FVC, decrease in FEV_1, decrease in FEV_1/FVC.



Patients with COPD and IC/TLC of less than or equal to 0.25 have an impaired LV diastolic filling and impaired global RV function.

ECHO:

LVDD is confirmed by[28,29,30]

1. LVEF greater than 50%
2. LV end diastolic volume index less than 97 mL/m^2
3. Invasively measured LV end diastolic pressure greater than 60 mmHg
4. Mean Pulmonary Capillary Wedge Pressure greater than 12 mmHg
5. LV chamber relaxation greater than 48 ms
6. Constant of LV chamber stiffness greater than 0.27

Global RV function is assessed by Tei's index or Myocardial performance index

Tei's index = Isovolumetric contraction time + Isovolumetric relaxation time/ Ejection time.

It is increased in COPD with pulmonary hypertension.

Miller manoeuvre during 2D ECHO study of normal persons shows increased right ventricular loading and acute leftward displacement of inter ventricular septum during end diastole end systole.

The correlation between LA and LV filling performance with RV pressure and diameter is called as Ventricular Interdependence.

ECHO in COPD patients show inspiratory drop in pleural pressure. Increased transmural pressure of LV increases LV afterload and LV Ejection Fraction.

COPD and skeletal muscle wasting

There is decrease in muscle mass and strength in COPD patients[31] due to

1. Deconditioning
2. Disuse atrophy
3. Systemic inflammation induced protein metabolism
4. Oxidative stress malnutrition

Increase in TNF α decreases muscle mass, causes loss of type II A muscle fibers[32]. Systemic oxidative stress also causes the same[33].

Ubiquitin proteasome activation causes active proteolysis and muscle wasting[34].

COPD and Cachexia

Leptin causes activation of sympathetic nervous system, vascular smooth muscle hypertrophy and cardiovascular remodelling. Adiponectin has anti leptin effects and is protective. Cachexia in COPD causes impaired production of adiponectin and increase leptin effects[35,36].

COPD and Osteoporosis

Increased prevalence of osteoporosis in COPD patients causes increased incidence of vertebral compression fractures. The inflammatory mediators, TNF α, IL 1β, IL 6 activate osteoclast and promote bone resorption[37].

Prolonged use of steroids results in reduced Osteoprotegrin production and upregulates RANK 1 ligand which in turn causes osteoclast activation resulting in bone resorption[38]. Inflammatory cytokines also reduce vitamin D levels in COPD patients adding to osteoporosis.

COPD and Depression

Depression in COPD patients is associated with increased IL 6. This is due to loss of independence with increase in disability. Cognitive dysfunction also occurs in COPD[39,40].

COPD and anaemia:

Normocytic normochromic anaemia occurs in COPD. Inflammatory cytokines produced in COPD patients reduce RBC survival and increase erythropoietin levels. Conversely, anaemia occurs due to peripheral erythropoietic resistance[41].

COPD and lung cancer:

Risk of lung cancer is increased in COPD due to chronic inflammation with increased production of growth factors and angiogenic factors like nuclear factor erythroid 2-related factor 2 (Nrf2)[42].

COPD and Metabolic syndrome

TNF α, IL-6 induce insulin resistance and results in metabolic syndrome in COPD patients[43,44].

Treatment of comorbidities

The primary reason for comorbidities in COPD is pulmonary inflammation. Treatment of comorbidities may be divided into two types

1. Treatment of comorbidities with routine COPD therapy
2. Treatment of comorbidities per se

Treatment of comorbidities with routine COPD therapy

The available modalities are

1. Inhaled Corticosteroids
2. Bronchodilators
3. Lung volume reduction surgery
4. Oxygen therapy
5. Pulmonary rehabilitation

Inhaled Corticosteroids reduces all-cause mortality in patients with COPD[45], but there is evidence that ICS either alone or used with LABA (long acting β2 agonist) has no role in suppressing systemic inflammation associated with COPD[46].

LABA, though no role in inflammation suppression improves skeletal muscle mass and prevents fatigue in COPD patients[47]. Anti cholinergics reduce inflammation mediated through mechanical forces of the lung causing mechanical strain to epithelial cells[48]. Theophylline in low doses reduce inflammation mediated by neutrophils[49].

Lung volume reduction surgery improves BMI, metabolic profile, osteoporosis, cardiac function, BODE index and long term survival[50,51]

Continuous home oxygen therapy and pulmonary rehabilitation improves exercise endurance, BODE scores and survival in COPD patients[52].

Treatment of comorbidities

This includes

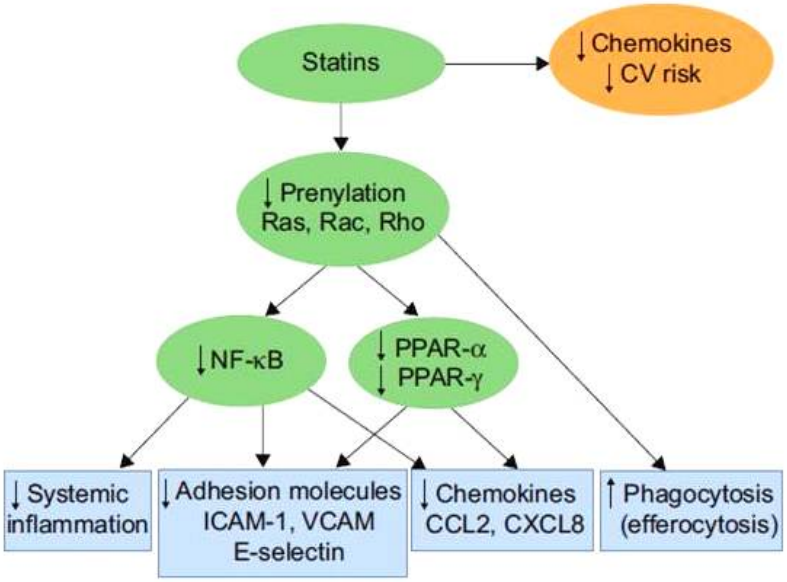
1. Statins
2. ACE inhibitors
3. PPAR agonists

Statins despite reducing total cholesterol levels, also have anti-oxidant, anti-inflammatory, and immune modulatory effects[53,54].

Statins are used in comorbidities like cardiovascular diseases, diabetes, osteoporosis and lung cancer[55].

The effects mediated by statins are shown in figure.

BENEFICIAL EFFECTS OF STATINS IN REDUCING INFLAMMATION IN COPD

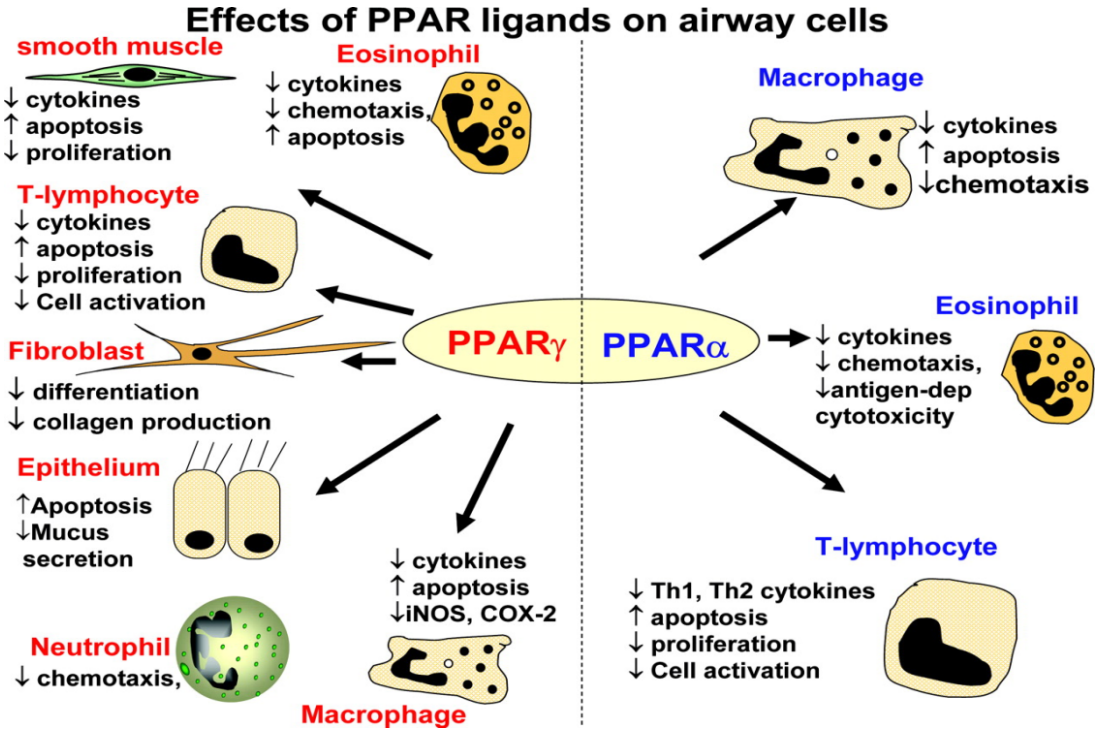


ACE inhibitors used in heart failure and hypertension reduces inflammatory effects mediated through angiotensin II[56].

Reduced expression of PPAR - α and PPAR - γ mRNA is found in COPD patients with cachexia.

PPAR agonists pioglitazone is useful in cachexia, skeletal muscle weakness and reducing systemic inflammation[57,58].

They have both anti-inflammatory and immuno modulatory effects.



Newer Therapies Under Research

1. Phospodiesterase 4 inhibitors

2. P38 Mitogen activated protein kinases

3. NF-kB inhibitors

MATERIALS AND METHODS

Setting

The study was conducted in Government Mohan Kumaramangalam Medical College, Salem.

Study Design:

Cross- sectional study.

Period of Study:

July 2010 to June 2012

Sample Size:

50 Cases; 40 Controls

Study Population:

Patients admitted in medicine wards of Government Mohan Kumaramangalam Medical College, Salem.

Ethical Committee Clearance: Obtained

Conflict Of Interest: Nil

Financial Support: Nil

Case Definition:

The cases in this study have following characters:

- Cases presenting with symptoms suggestive of chronic airway obstruction like cough, cough with expectoration of sputum of more than 2 years duration, dyspnoea, and with (or) without swelling of both legs.
- Cases in whom clinical diagnosis of COPD was made.
- All the cases were subjected to spirometry and the presence of COPD was confirmed by post bronchodilator spirometry values of

i. $FEV_1 < 80\%$.

ii. $FEV_1 / FVC < 0.7$.

iii. Reversibility of obstruction < 15%.

(FEV_1 – Forced Expiratory Volume in 1 second FVC – Forced Vital Capacity)

Inclusion Criteria:

- Patients with age greater than 35 years
- Patients with clinical and spirometric evidence suggestive of COPD

Exclusion Criteria:

- Patients with other respiratory diseases like:
    a. Asthma,
    b. Tuberculosis,
    c. Bronchiectasis,
    d. Lung malignancy
- Patients with Cardiac diseases like:
    a. Valvular heart diseases,
    b. Coronary heart diseases,
    c. Hypertension,
    d. Cardiomyopathies

- Patients with AIDS
- Patients who cannot perform Spirometry or with poor echo window

Controls:

Age matched, non smoking, healthy volunteers.

Methods:

Both cases and controls were subjected to :

1) History taking and clinical examination.
2) Routine blood and urine investigations
3) Sputum AFB
4) HIV Serology
5) Chest X-Ray PA View
6) Electrocardiography
7) Echocardiography
8) Spirometry

Spirometry

Spirometry is a method of assessing lung function by measuring the volume of air that the patient can expel from the lungs after maximal inspiration.

The indices derived from forced exhaled manoeuvre have become the most accurate and reliable way of supporting a diagnosis of COPD.

When these values are compared with predicted normal values determined on the basis of age, height, sex, and ethnicity, a measure of the severity of airway obstruction can be determined. Based on these values COPD has been classified as mild, moderate, severe and very severe disease levels.

Its major uses in COPD are to:

1. Confirm the presence of airway obstruction
2. Confirm FEV_1/FVC ratio of < 0.7 after bronchodilator
3. Provide an index of disease severity
4. Help differentiate asthma from COPD

5. Detect COPD in subjects exposed to risk factors, predominantly tobacco smoke independent of the presence of respiratory symptoms

6. Enable monitoring of disease progression

7. Help assess response to therapy

8. Aid in predicting prognosis and long-term survival

9. Exclude COPD and prevent inappropriate treatment, if spirometry is normal

Procedure of Spirometry

1. Maximal inspiration

2. Maximal expiration ("blast" expiration)

3. Continued expiration until maximal amount of air is exhaled (residual volume) (at least a 6 second exhalation in adults)

4. Get 3 readings –within 5% or 100ml of each other.

5. Use best values for FEV_1,FVC and FEV_1/ FVC Ratio.

Information Provided by the Spirometer

- ***FEV_1 (Forced expiratory volume in one second)***: The volume of air exhaled in the first second of the blow.

- ***FVC (Forced vital capacity)***: The total volume of air that can be forcibly exhaled in one breath.

- ***FEV_1/FVC ratio***: The fraction of air exhaled in the first second relative to the total volume exhaled.

- The ratio of FEV_1/FVC is normally between 0.7 and 0.8. Values below 0.7 are a marker of airway obstruction, except in older adults where values of 0.65–0.7 may be normal.

Bronchodilator Reversibility Testing in COPD

Assessing bronchodilator reversibility is important to determine whether fixed airway narrowing is present. In patients with COPD, post-bronchodilator FEV_1/FVC remains < 0.7. However, the FEV_1 may improve significantly after bronchodilator, and a change of > 12% or > 200 mL in FEV_1 can occur in COPD.

1. Spirometry should be undertaken when the patient is clinically stable and free from a respiratory tract infection.

2. Short-acting bronchodilators should be withheld for the previous 6 hours, long-acting bronchodilators for 12 hours, and sustained release of theophylline for 24 hours.

3. FEV_1/FVC should be measured before and 15-20 minutes after bronchodilator is given.

4. The bronchodilator should be given by metered dose inhaler, ideally through a spacer. A nebulizer may be used but generally larger doses are delivered by this route.

5. The dose administered should be high on the dose-response curve.

6. Possible dose protocols include 400 μg salbutamol, up to 160 μg ipratropium, or the two combined.

Calculating bronchodilator reversibility:

% FEV_1 Reversibility =

$$\frac{\textit{Post-bronchodilator } FEV_1 - \textit{Pre bronchodilator } FEV_1}{\textit{Pre-bronchodilator } FEV_1} \times 100$$

Contraindications:

Increases Intraocular, Intrathoracic, Intra-abdominal and Intracranial pressure

1. At least 6 weeks since the last exacerbation.
2. Recent MI less than 3-6 months ago.
3. Unstable angina in last 24 hours.
4. Haemoptysis of unknown origin.
5. Recent eye surgery less than 3-6 months.
6. Abdominal surgery within last 3-6 months.
7. Recent CVA less than 3-6 months.
8. Diagnosis of Tuberculosis unless special precautions used.
9. Current chest infection or within last 6 weeks.
10. Current chest pain with no diagnosis.
11. Pulmonary embolism (PE) within last 3-6 months.
12. Ear infection.

13. Spontaneous pneumothorax.

14. Aortic aneurysm.

GOLD COPD Staging

GOLD divides COPD into four stages based on FEV_1% and FEV_1/FVC% determined by spirometry

1. Mild

2. Moderate

3. Severe

4. Very severe

GOLD COPD STAGING

Stage	Description	Findings (based on postbronchodilator FEV1)
0	At risk	Risk factors and chronic symptoms but normal spirometry
I	Mild	FEV1/FVC ratio less than 70 percent FEV1 at least 80 percent of predicted value May have symptoms
II	Moderate	FEV1/FVC ratio less than 70 percent FEV1 50 percent to less than 80 percent of predicted value May have chronic symptoms
III	Severe	FEV1/FVC ratio less than 70 percent FEV1 30 percent to less than 50 percent of predicted value May have chronic symptoms
IV	Very severe	FEV1/FVC ratio less than 70 percent FEV1 less than 30 percent of predicted value or FEV1 less than 50 percent of predicted value plus severe chronic symptoms

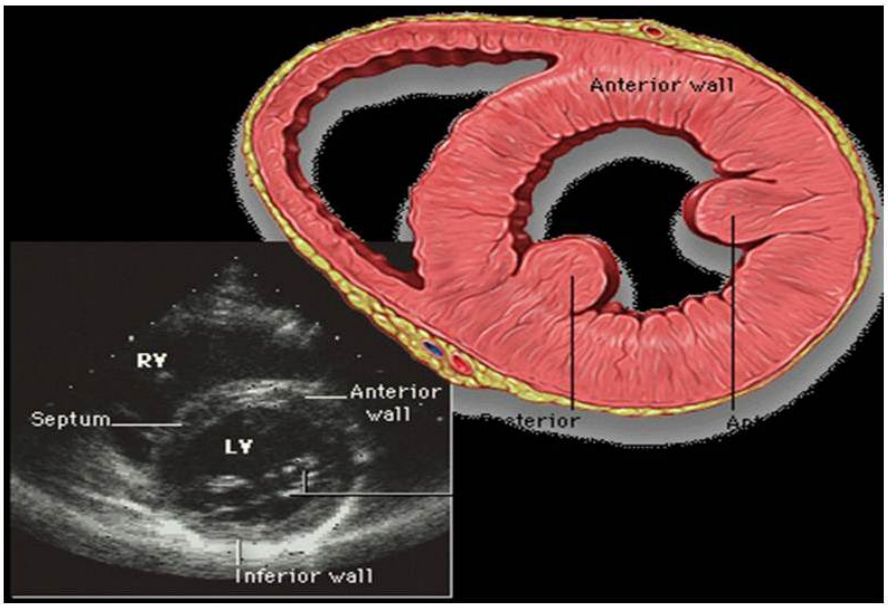
Echocardiography:

The Doppler echocardiographic examinations were performed using a commercially available Doppler echocardiograph and a transducer array of 2.5 MHz. Images were obtained from parasternal views (long axis and short axis) and apical four-chamber view.

The subjects were placed in left lateral decubitus position for the parasternal views and in a supine position for the apical four-chamber view. Subjects stay at rest for 10 minutes before the Echocardiographic examination.

Right Ventricular Enlargement

APICAL TWO CHAMBER VIEW SHOWING BOTH VENTRICULAR CAVITIES



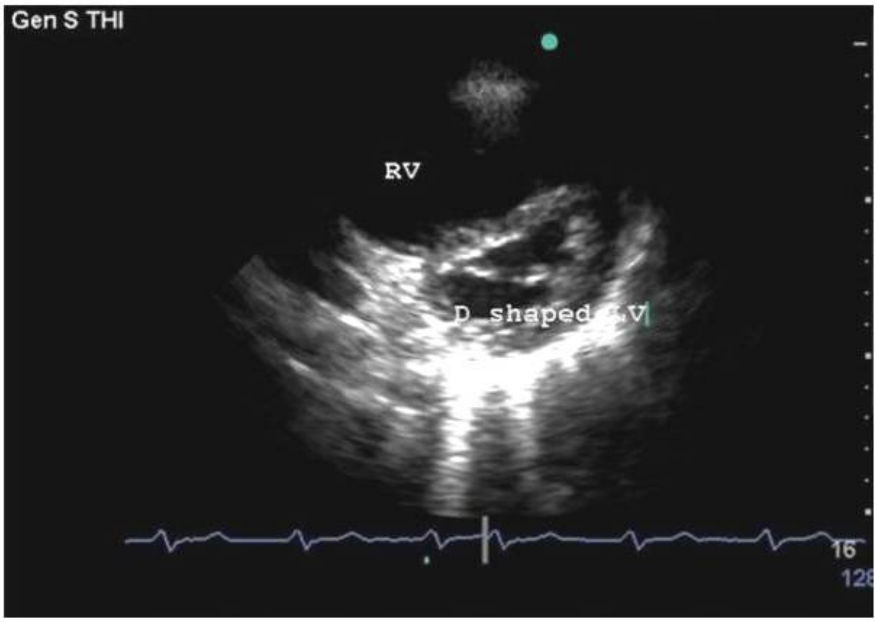
Parasternal short axis view is used to determine RV enlargement. In this view, RV is thin with a cresentric cavity anterior to LV. LV cavity is usually rounded. RV cavity is usually smaller than that of LV in cross section.

When RV dilates or hypertrophies :

1. RV cross section is greater than LV cross section

2. LV cavity becomes D- shaped rather than rounded

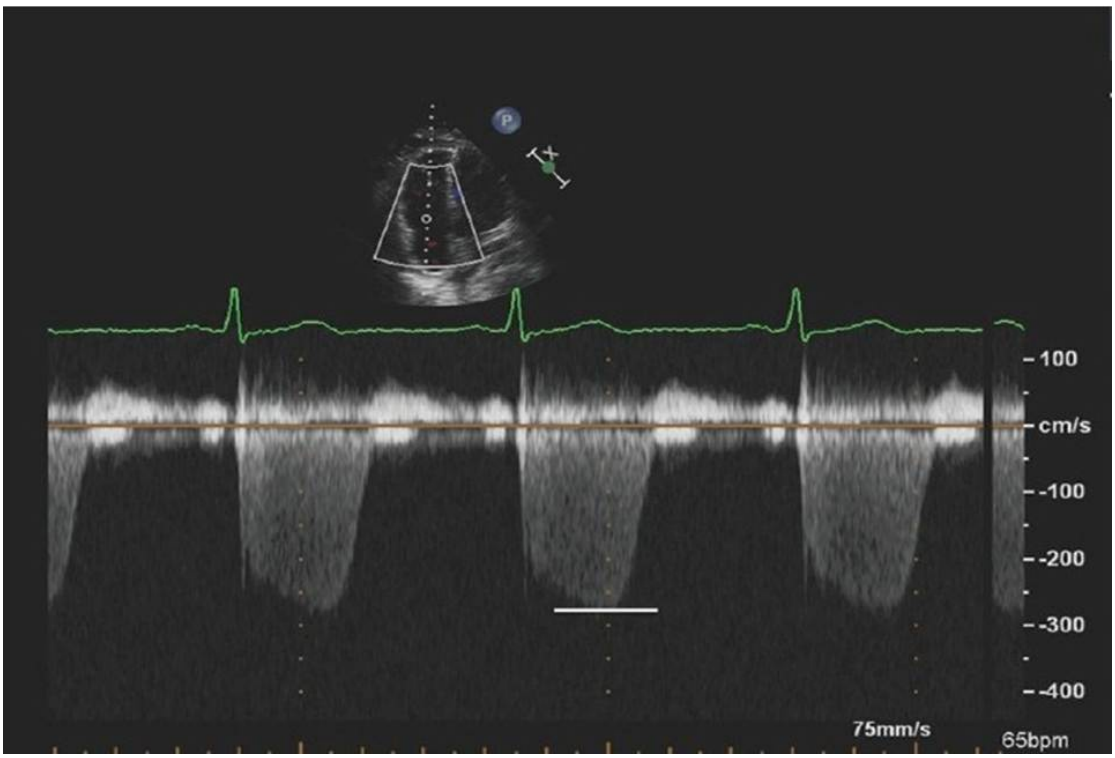
IMAGE SHOWING RV DILATION



Measurement of Systolic Pulmonary Arterial Pressure

Systolic PAP is equal to right ventricular systolic pressure(RVSP) in the absence of right ventricular outflow tract obstruction or pulmonary stenosis.

Tricuspid regurgitant jet velocity in apical four chamber view



RVSP = Tricuspid pressure gradient(TVpg) + Right atrial pressure(RAP)

TVpg is the pressure gradient across both RA and RV during systole and is measured by Bernoulli's equation.

Bernoulli equation, TVpg = $4v^2$

'v' is the tricuspid regurgitant jet velocity measured in apical 4 chamber view by placing color wave doppler line placed in the centre of the jet.

Right atrial pressure is assessed by Caval index, IVC collapse with respiration. (Complete collapse – 5 mm Hg, Partial collapse – 10 mm Hg, No collapse – 15 mm Hg).

LV systolic function

Standard indices of global LV systolic performance are

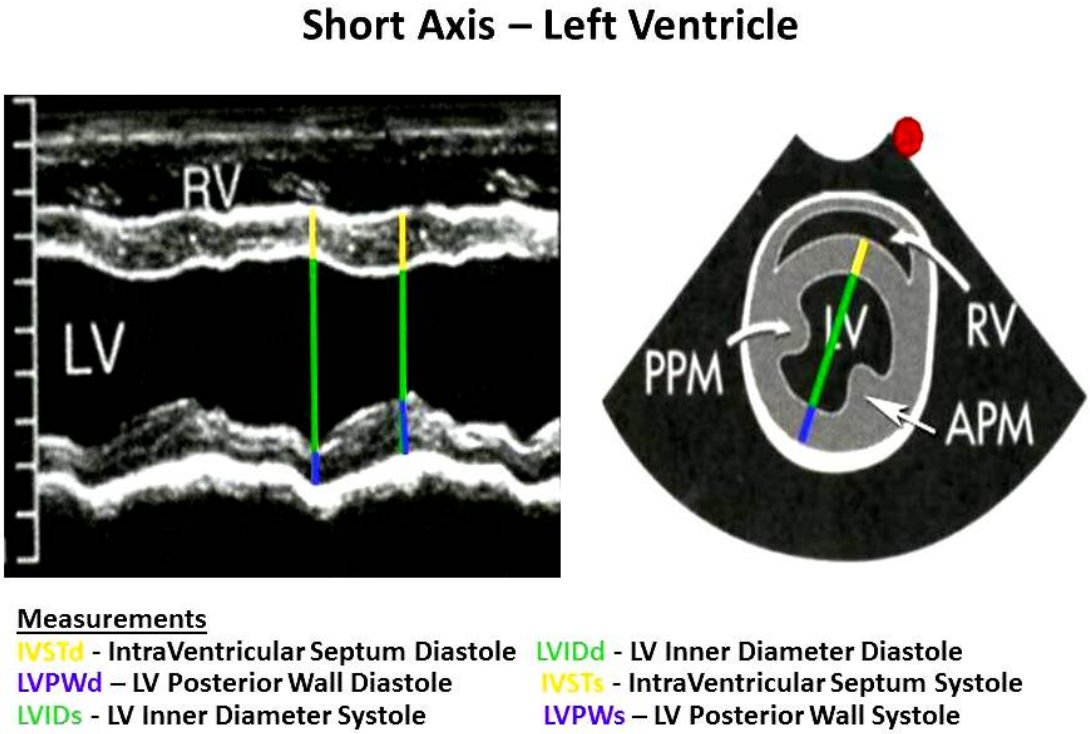
1. Ejection Fraction (EF) and
2. LV percent fractional shortening (%FS).

These indices are usually estimated from various cardiac diameters.

1. Left ventricle end-systolic and end diastolic diameters (LVESD, LVEDD),
2. Inter ventricular septal thickness (IVS),
3. Right ventricle end-systolic and end diastolic diameters (RVESD, RVEDD)

4. Left ventricular posterior wall thickness at end diastole and end systole.

The above indices were measured by M-mode echocardiography from the left parasternal short- and long-axis views.



%FS was taken as the ratio of (LVEDD – LVESD)/LVEDD

EF was determined using the Teichholz formula.

LV diastolic function.

The indices used for assessment of LV diastolic function are :

Mitral inflow patterns

1. E/A ratio,

2. Deceleration time,

3. Iso volumetric relaxation time (IVRT)

4. Mitral annulus velocities on tissue doppler: E/e' ratio

The flow from the left atrium to the left ventricle occurs in 3 phases:

I. Initial rush of blood as soon as the valve opens causes peaking of velocity in early diastole, the ’E‘ wave.

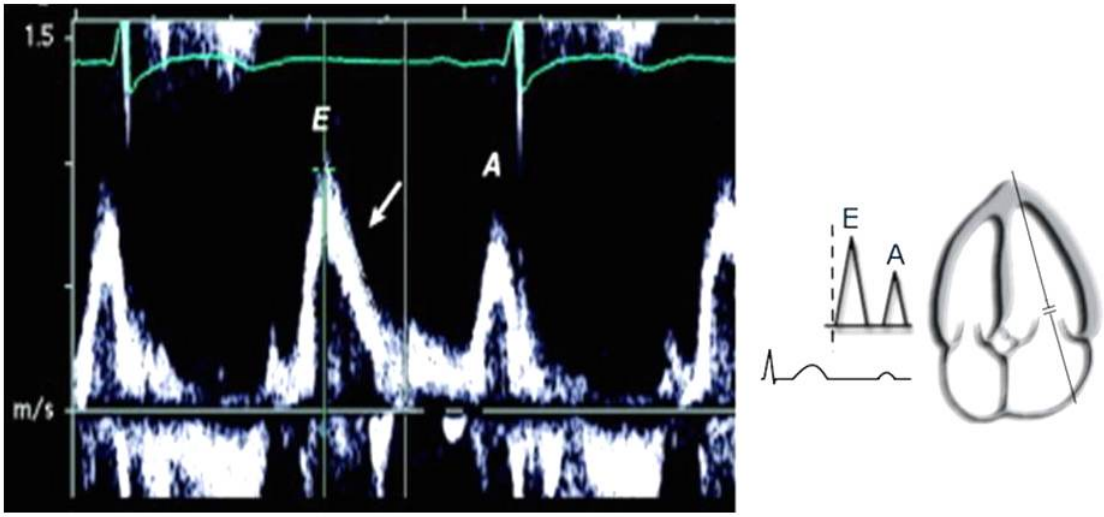
II. This is followed by a period of low or no flow, also known as diastasis.

III. In end-diastole, atrial contraction produces a final rush of blood into the ventricle, the ’A’ wave.

This was measured by placing the cursor over the tips of open mitral leaflets in apical 2 chamber or 4 chamber view.

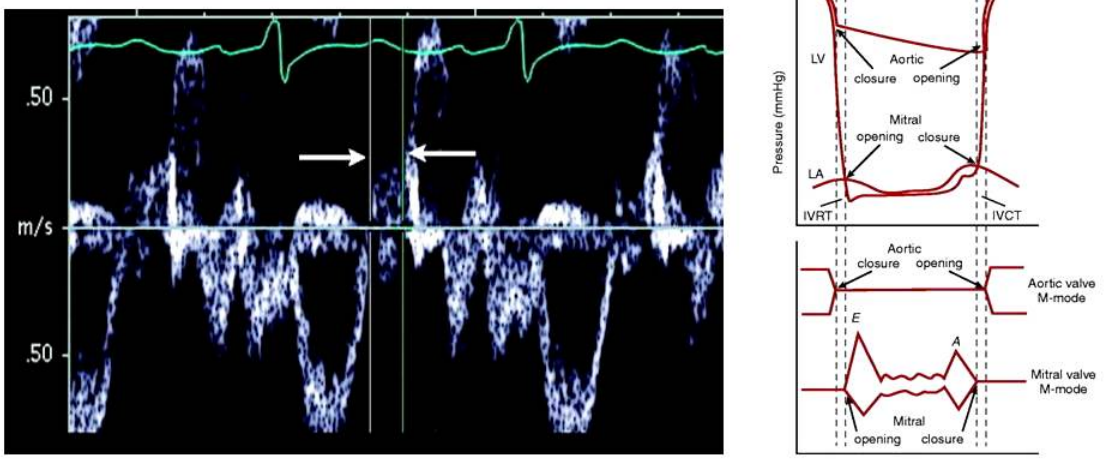
The deceleration time (DT) is the time taken from the maximum E point to baseline.

TRANS MITRAL INFLOW



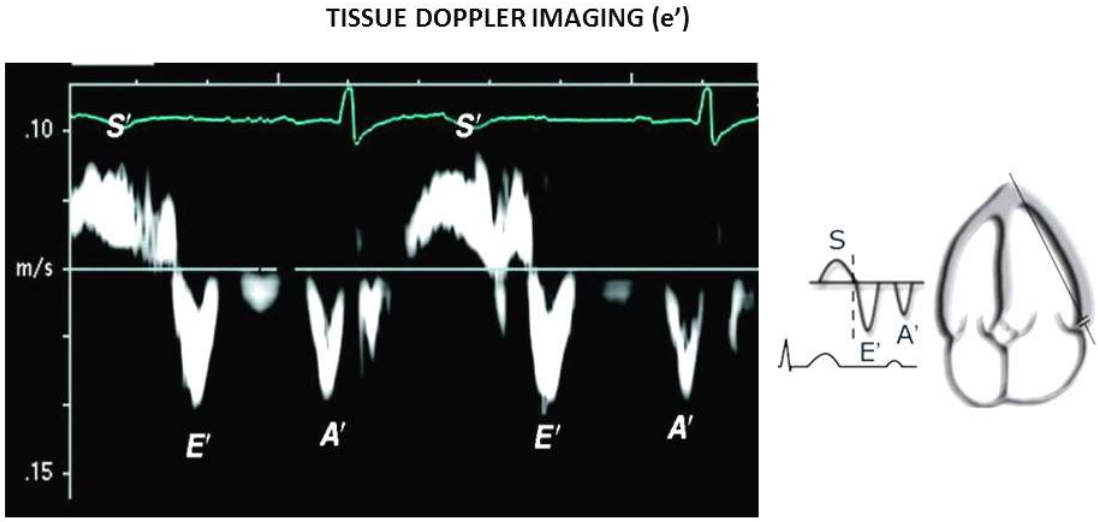
IVRT is measured as the time between the closure of the aortic valve and the opening of the mitral valve.

IVRT



In apical 4 chamber view, in tissue Doppler imaging, a 2mm to 5mm sample volume is placed over the medial mitral annulus, at the base of the mitral leaflet. Typically, two negative waves in diastole and one

positive wave in systole is seen. The first of the diastolic waves is the result of movement of the annulus towards the left atrium during initial filling of the LV. This wave is referred to as e'. The second diastolic wave is referred to as a'. The systolic wave is labeled as s'.



Diastolic filling abnormalities can be graded as follows:

Grade 0: Normal diastolic function (E > A)

Grade 1: Impaired relaxation (E:A reversal i.e. E is < A)

Grade 2: Pseudonormal (E:A ratio appears normal)

Grade 3: Restrictive filling (E:A ratio often > 2)

Statistical Analysis

The data collected were scored & analysed using SPSS (Statistical Package for Social Science) Ver 16.01. Continuous variables were presented as Mean (M) with Standard deviation (sd) and categorical variables were presented as frequency and percentages. Student t-test and analysis of variance (ANOVA) were used for testing the significance of all the variables (Mean & sd) in both groups. Chi-square test was used to compare proportions. All the statistical results were considered significant at P value < 0.05.

OBSERVATION AND RESULTS

Table - 1 : Sex distribution of the Sample

Sex	CASE N=50		CONTROL N=40	
	N	**%**	**N**	**%**
Male	28	56.00	20	50.00
Female	22	44.00	20	50.00
Chi-square value	**0.32**			
D_f	**1**			
p-value	**0.57 (Not Significant)**			

From the above table it is observed that, number of males is slightly higher than females in the study group.

But there was no significant difference in sex between the study group and the control group.

Chart - 1 : Sex distribution of the Sample

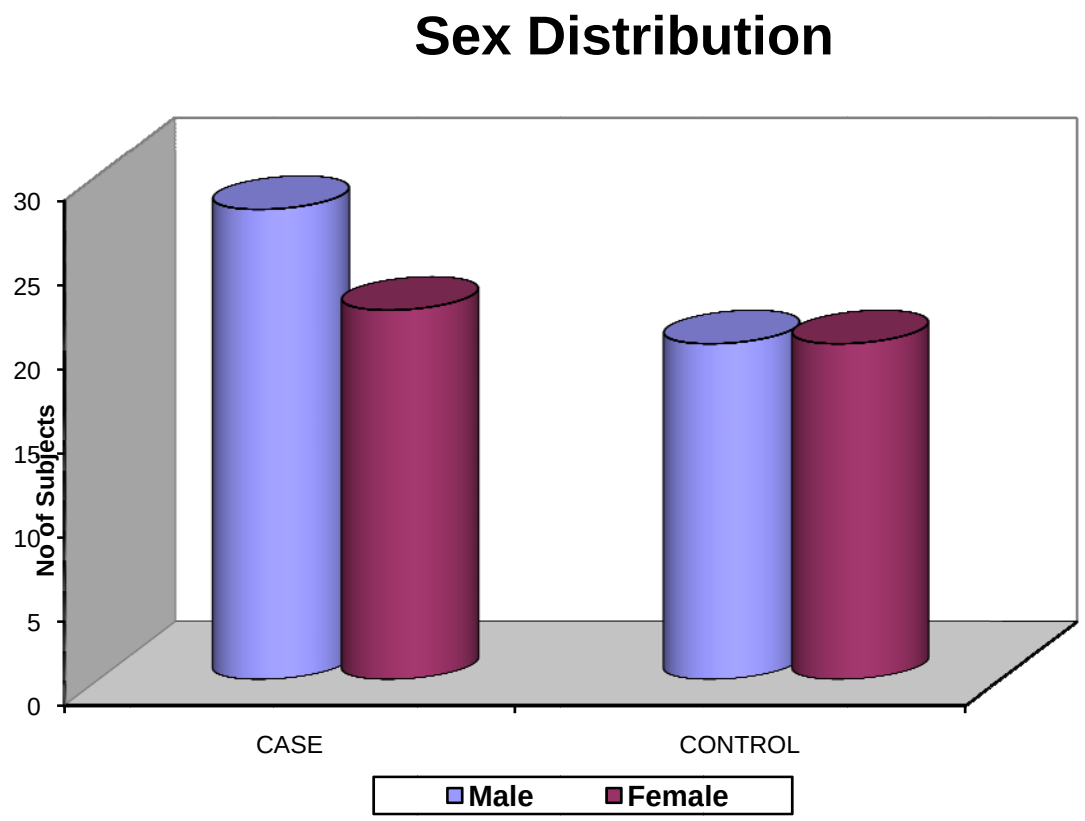


Table - 2 : Age Distribution of the Study Sample

	CASE N=50		CONTROL N=40	
	N	**%**	**N**	**%**
35-45	9	18.00	10	25.00
45-55	28	56.00	11	27.50
55-65	11	22.00	14	35.00
65-75	2	4.00	2	5.00
75-85	0	0	3	7.50
Mean (sd)	**51.94 ± 7.57**		**55.20 ± 11.34**	
T-value D_f p-value	**1.63** **88** **0.11 (Not Significant)**			

There is no significant difference in age between COPD and control group. The minimum age of the patient was considered to be 35 because COPD is usually a disease of middle age and is less likely below 35 years.

Chart - 2 : Age Distribution of the Study Sample

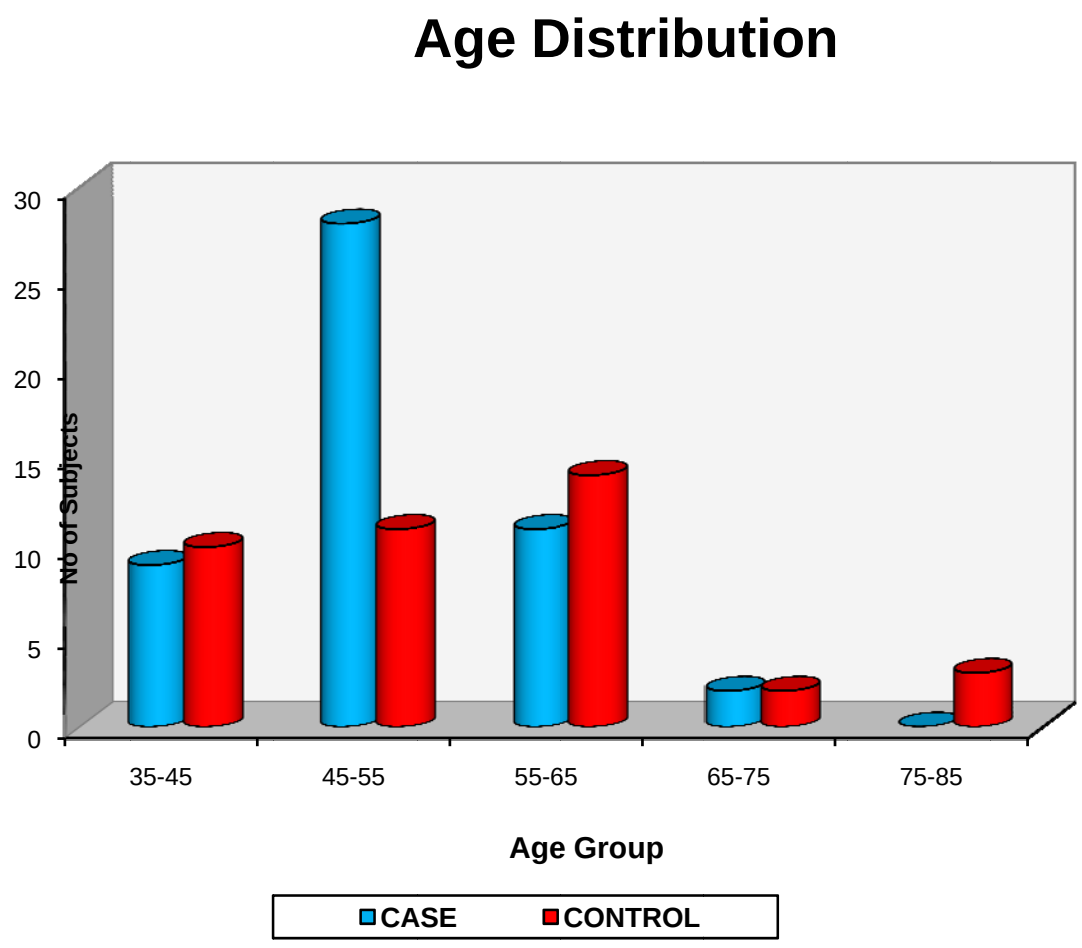


Table-3 : FEV_1

	CASE	CONTROL
Mean	0.98	3.15
Sd	0.28	0.60
t-Value	**22.73**	
Df	**88**	
p-value	**0.000 (Significant)**	

From the above table it is inferred that mean difference in FEV1 in the study group is 0.98±0.28 and in control group is 3.15± 0.60.

By applying student t test, p value was found to be significant between the two groups.

Table - 4 : FVC

	CASE	**CONTROL**
Mean	1.92	3.86
sd	0.28	0.75
t-Value	**16.76**	
D_f	**88**	
p-value	**0.000 (Significant)**	

From the above table, it is observed that mean difference in FVC is 1.92±0.28 in COPD group and 3.86±0.75 in the control group.

P value was significant between the two groups.

Table - 5 : FEV_1/FVC

	CASE	**CONTROL**
Mean	51.10	81.83
sd	7.94	4.44
t-Value	**21.86**	
D_f	**88**	
p-value	**0.000 (Significant)**	

From the above table, it is inferred that mean difference in the FEV_1/FVC in COPD group was 51.10±7.94 and control group was 81.83±4.44. p value was significant.

Table - 6 : Corpul Monale

	Case		Control	
	Number	**Percentage**	**Number**	**Percentage**
Absent (A)	43	86.00	40	100
Presented (P)	7	14.00	0	
Total	50	100	40	100
Chi square	3.07			
D_f	1			
P value	0.01 (Significant)			

Statistically significant association was found in cor pulmonale in COPD group when compared to control group by applying chi square test.

Cor pulmonale was found in 14% of COPD patients

Chart - 6 : Corpul Monale

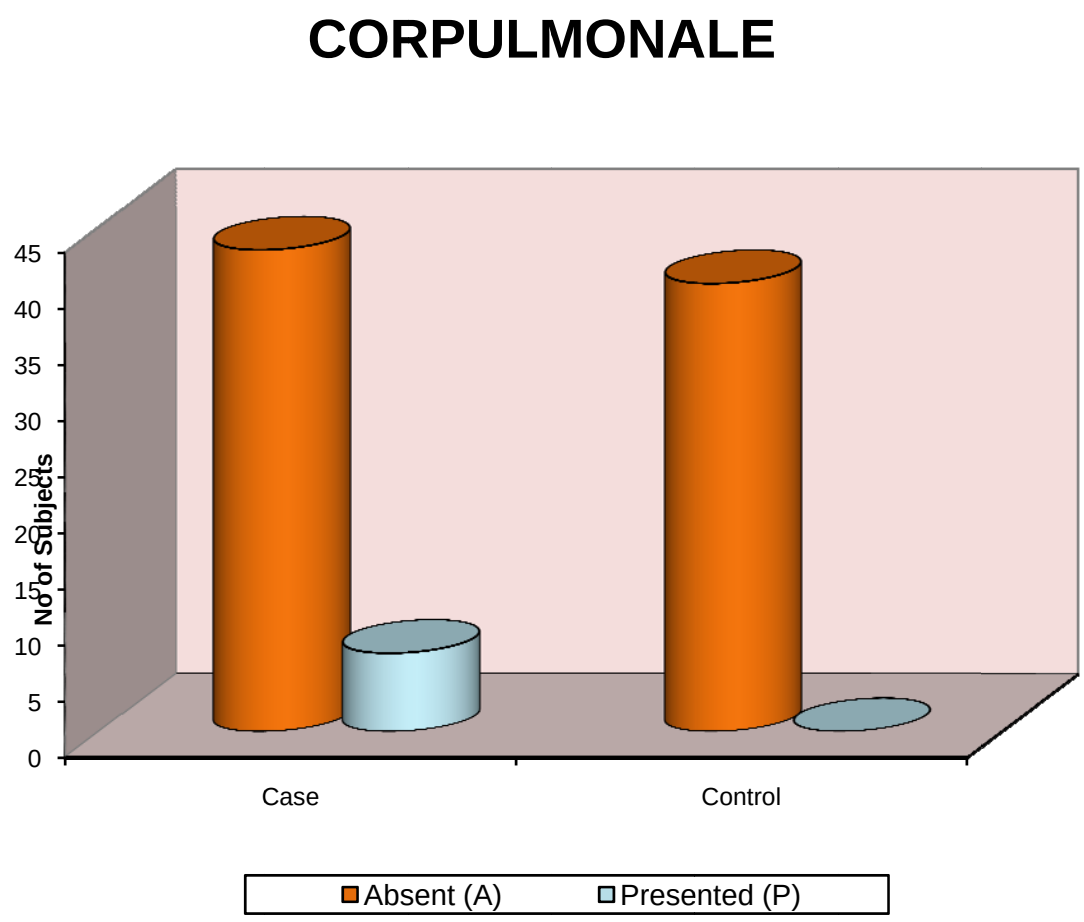


Table - 7 : Elevated PAP

	Case		Control	
	Number	**Percentage**	**Number**	**Percentage**
Absent (A)	31	62.00	40	100
Presented (P)	19	38.00	0	
Total	50	100	40	100
Chi square	19.27			
D_f	1			
P value	0.000 (Significant)			

Statistically significant association was found in elevated PAP in COPD group when compared to control group. PAP is elevated in 37% of the patients

Chart - 7 : Elevated PAP

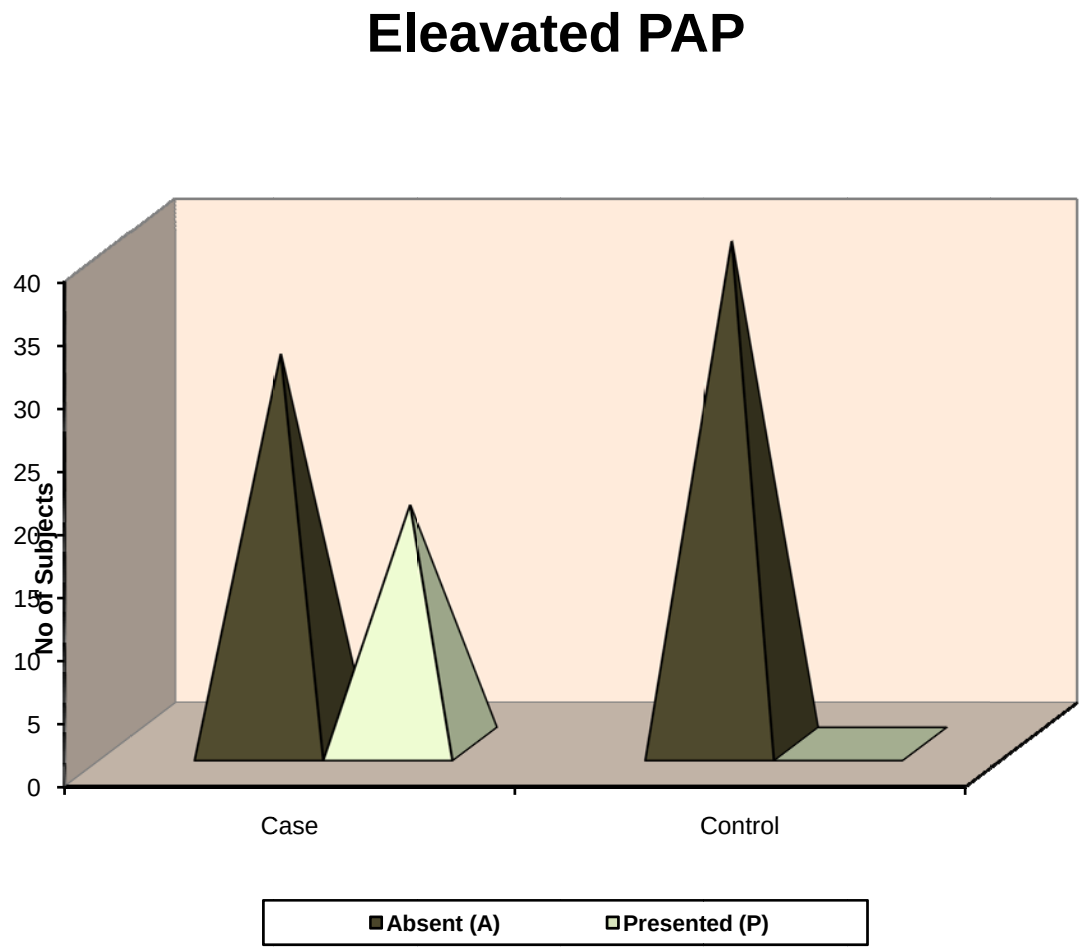


Table - 8 : LVDD

	Case		Control	
	Number	**Percentage**	**Number**	**Percentage**
Absent (A)	37	74.00	40	100
Presented (P)	13	26.00	0	0
Total	50	100	40	100
Chi square	12.16			
D_f	1			
P value	0.000 (Significant)			

Statistically significant association was found in LVDD in COPD group when compared with controls.

LVDD is found in 26% of the COPD patients

Chart - 8 : LVDD

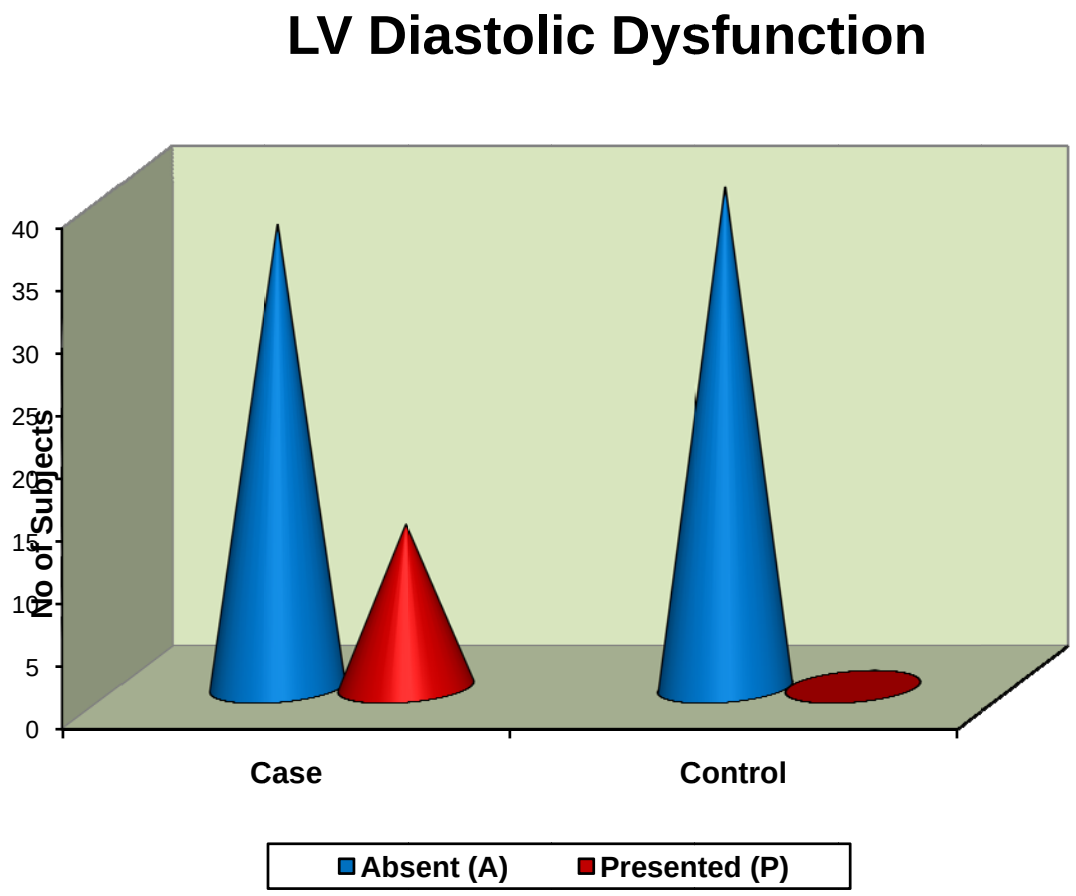

LV Diastolic Dysfunction
No of Subjects
40
35
30
25
20
15
10
5
0
Case
Control
Absent (A)
Presented (P)

Table - 9 : LVSD

	Case		Control	
	Number	**Percentage**	**Number**	**Percentage**
Absent (A)	50	100	40	100
Presented (P)	0	0	0	0
Total	50	100	40	100

No significant association was found in both COPD and control groups.

Chart - 9 : LVSD

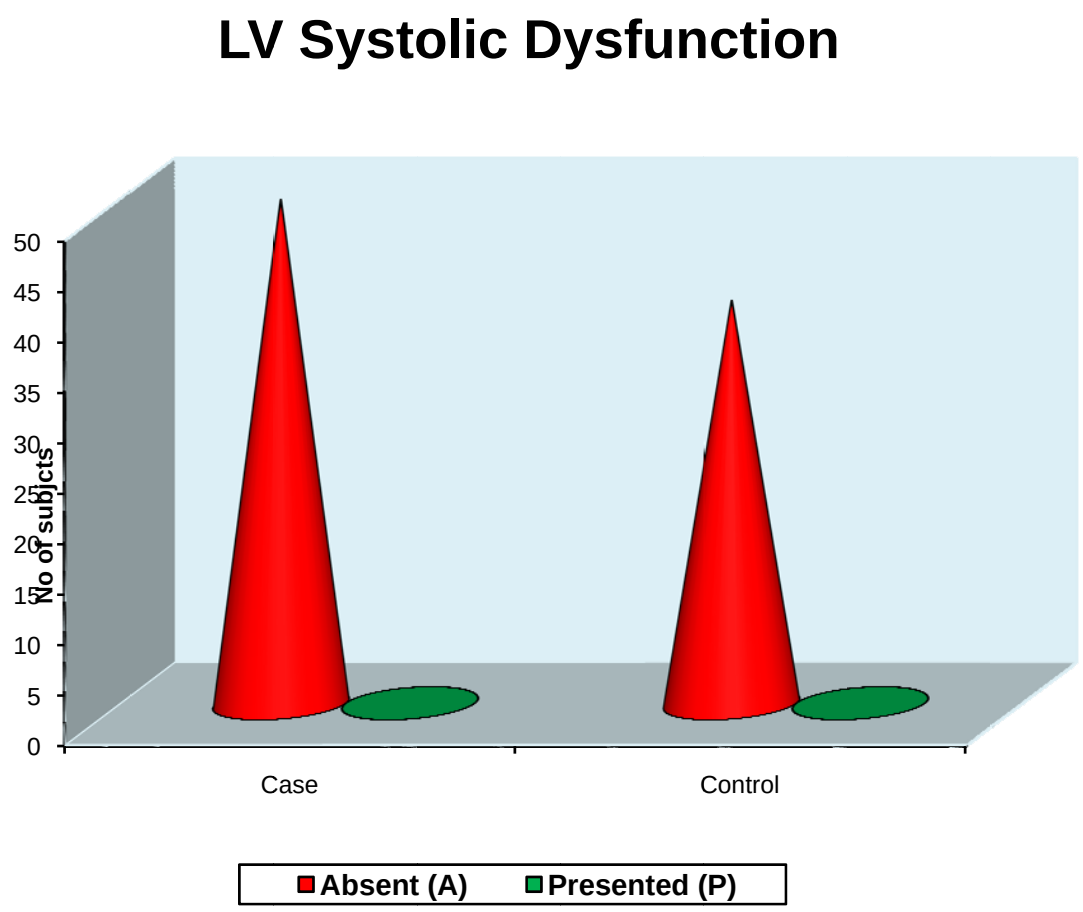

LV Systolic Dysfunction
No of subjcts
50
45
40
35
30
25
20
15
10
5
0
Case
Control
Absent (A)
Presented (P)

Table - 10 : GOLD COPD STAGING

	Number	**Percentage**
Mild	14	28.00
Moderate	26	52.00
Severe	6	12.00
Very Severe	4	8.00
Total	50	100

From the above table, it is observed that mean difference in the majority of patients in the COPD group were in the moderate group (FEV_1 50-80%) i.e. about 52%.

8% of patients showed very severe airflow obstruction i.e. GOLD stage IV (FEV_1<30%).

Table - 10 : GOLD COPD STAGING

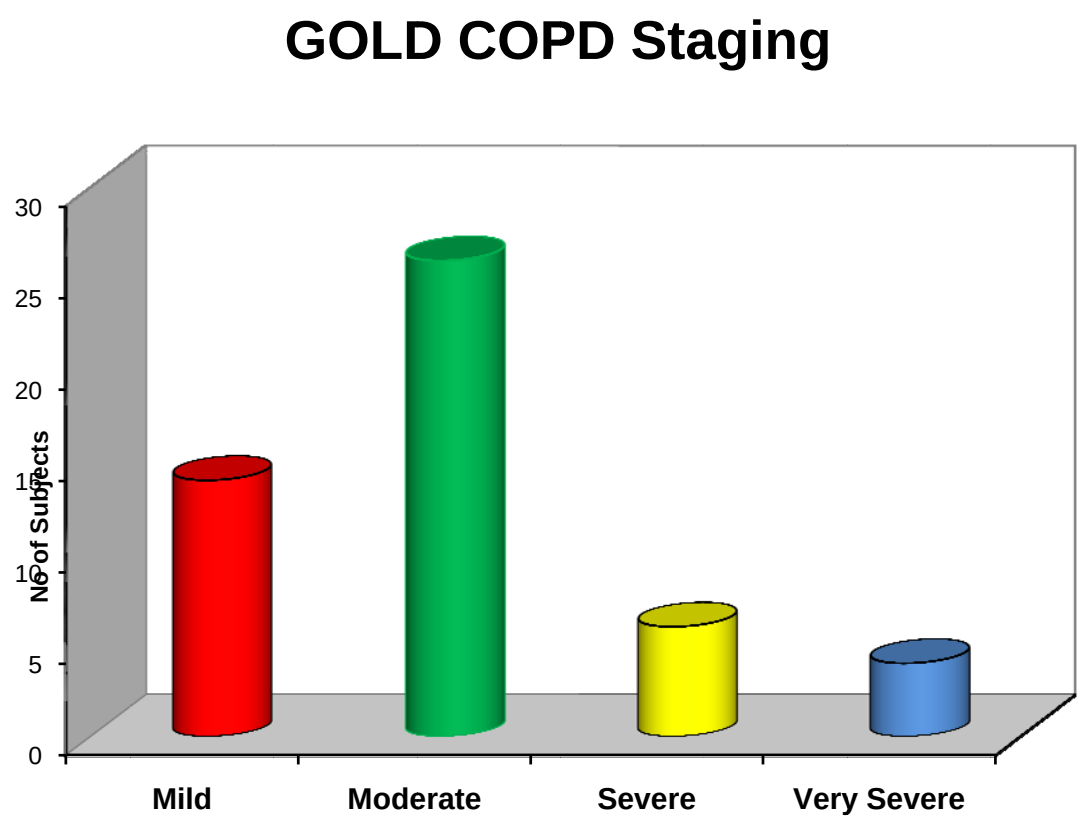


TABLE - 11 : GOLD COPD STAGING vs CORPUL MONALE

G C Staging	A		P	
	Number	Percentage	Number	Percentage
Mild	14	32.60	0	
Moderate	24	55.80	2	28.60
Severe	4	9.30	2	28.60
Very Severe	1	2.30	3	42.90
Total	43	100	7	100
Chi-square	17.36			
D_f	3			
P-Value	0.001 (Significant)			

From the above table, it is observed that there is significant association between GOLD COPD staging and cor pulmonale.

The association of cor pulmonale increases with increase in GOLD COPD staging.

TABLE - 11 : GOLD COPD STAGING vs CORPUL MONALE

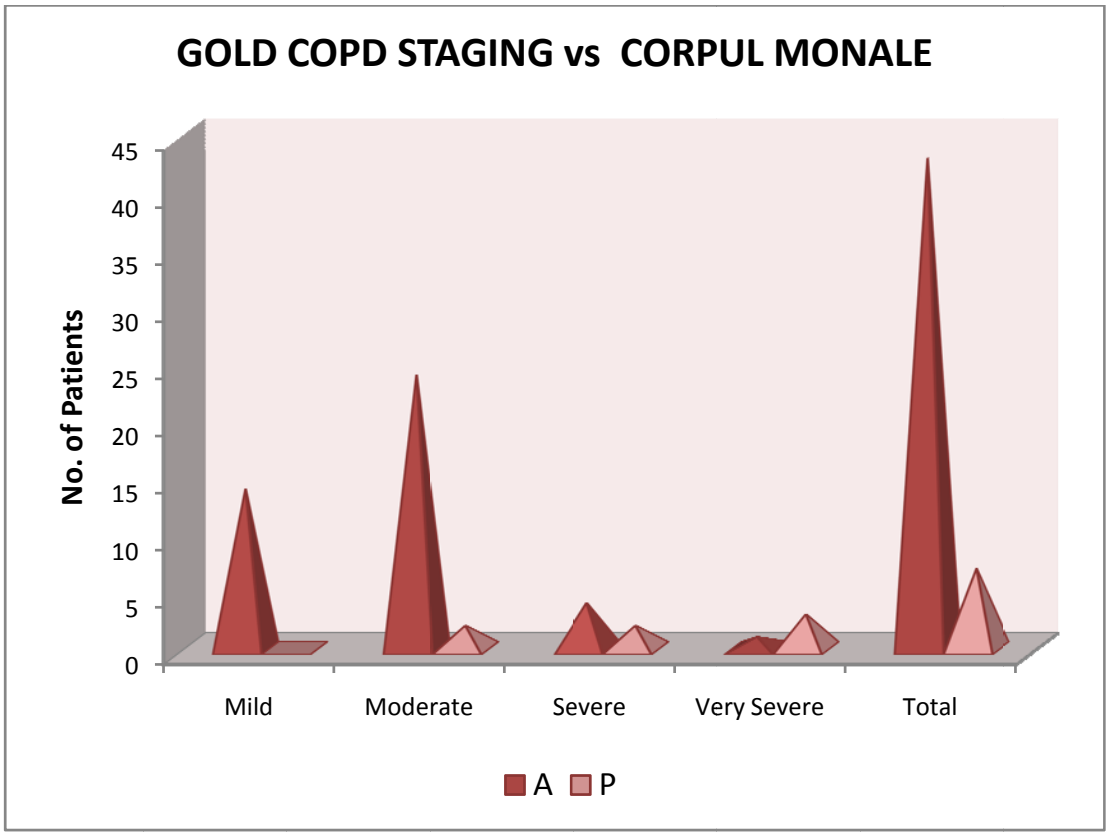


TABLE - 12 : GOLD COPD STAGING vs Elevated PAP

G C Staging	A		P	
	Number	**Percentage**	**Number**	**Percentage**
Mild	11	35.50	3	15.80
Moderate	18	58.10	8	42.10
Severe	02	6.50	4	21.10
Very Severe	0	0	4	21.10
Total	31	100	19	100
Chi-square	10.83			
D_f	3			
p-value	0.013			

From the above table, it is observed that there is significant association between GOLD COPD staging and elevated PAP.

Association of elevated PAP increases with increase in GOLD COPD staging.

Chart - 12 : GOLD COPD STAGING vs Elevated PAP

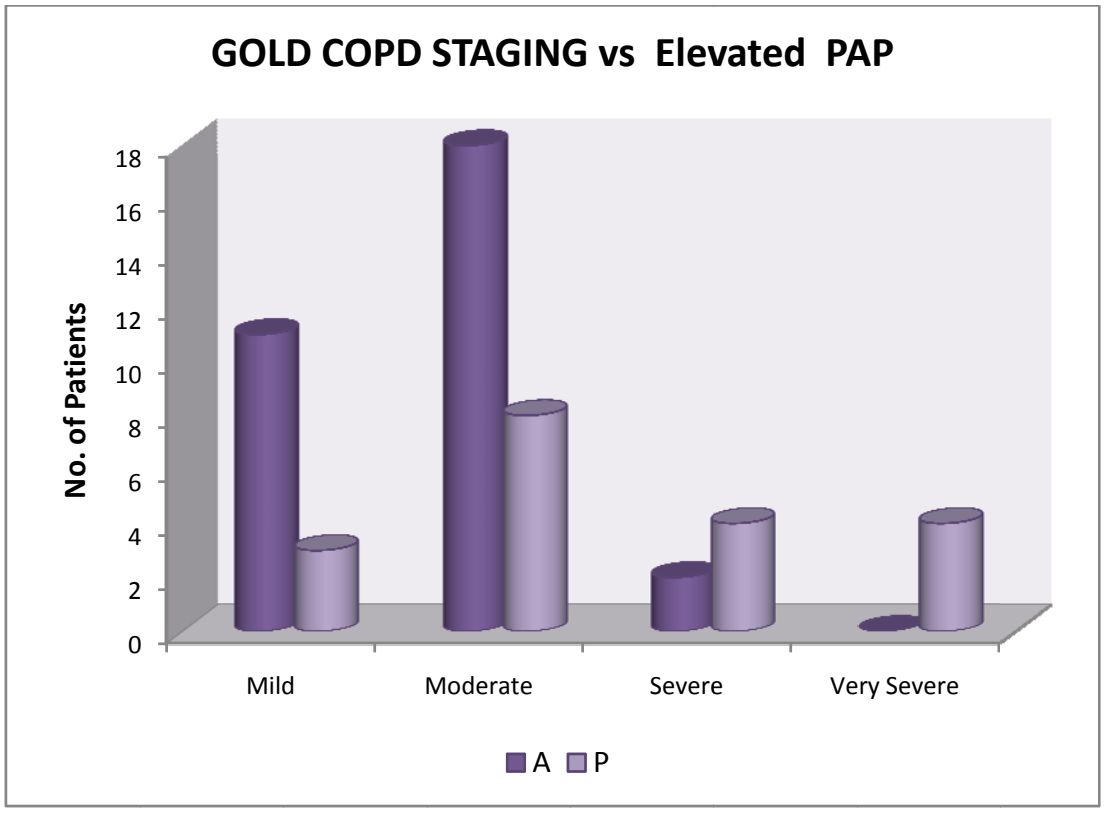


TABLE - 13 : GOLD COPD STAGING vs LVDD

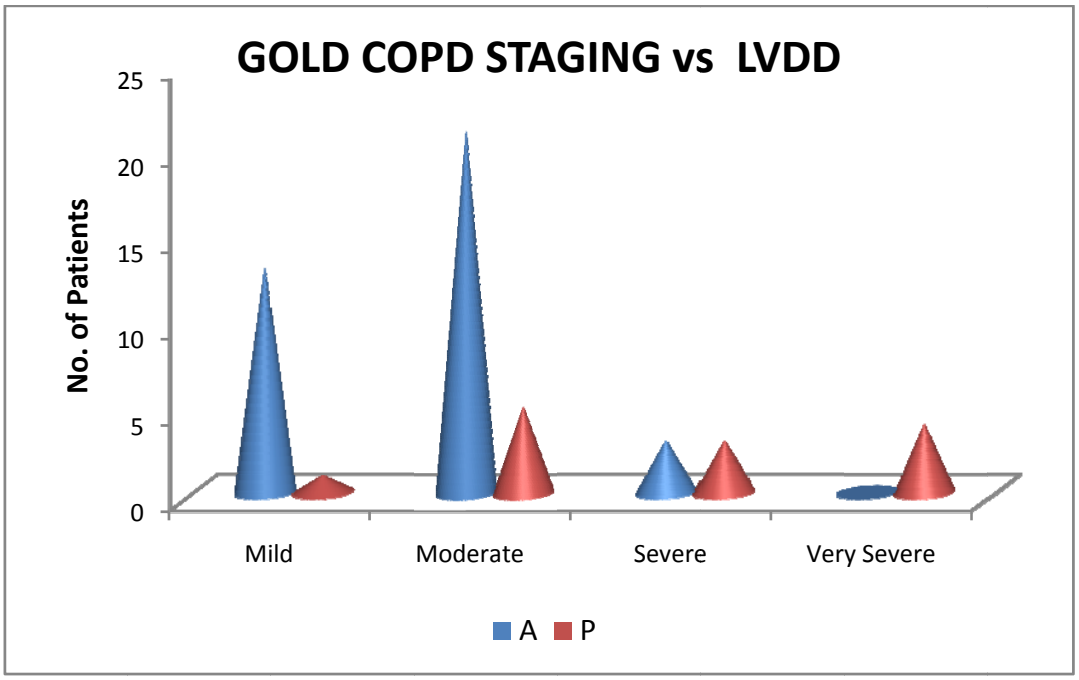
G C Staging	A		P	
	Number	Percentage	Number	Percentage
Mild	13	35.10	1	7.70
Moderate	21	58.00	5	38.50
Severe	3	08.10	3	23.10
Very Severe	0	0	4	30.80
Total	37	100	13	100
Chi-square	16.39			
D_f	3			
p- value	0.001 (Significant)			

From the above table, it is observed that there is significant association between GOLD COPD staging and LVDD.

LVDD can be seen in every stage of COPD.

Association of LVDD increases with increase in GOLD COPD staging.

Chart - 13 : GOLD COPD STAGING vs LVDD



Discussion

The common cardiac manifestations secondary to COPD are

1. Cor pulmonale
2. Elevated PAP
3. Ventricular dysfunction

Cor Pulmonale

Cor pulmonale is found in 14 percent of patients in this study. Rigolin et al[59] and Fishman et al[60] has found that in autopsy, cor pulmonale was found in 40 percent of COPD patients.

Elevated PAP

In this study, PAP is elevated in 38 percent of COPD patients, but the true prevalence of elevated PAP is variable, being about 20-90 percent, which was proved by various studies using right heart catheterization[61,62,63].

Elevated PAP is a prognostic indicator in COPD patients. Weitzenblum et al[64] has found that 5 year survival rate in patients with elevated PAP was 50 percent in mild PHT (20-30 mm Hg), 30 percent in

moderate to severe group (30-50 mm Hg) and 0 percent in very severe group (>50 mm Hg).

The frequency of elevated PAP in the study is 16 percent in mild, 42 percent in moderate, 21 percent in severe and 21 percent in very severe. Higham et al[65] has found it to be 25 percent, 43 percent and 68 percent in mild, moderate and severe group.

In our study, incidence of PHT increases with increase in COPD staging.

Right Ventricular Dysfunction

Bristow et al[66] described that right ventricle is thin-walled and eccentric, small increases in pulmonary artery pressure may result in large increases in right ventricular work and pulmonary hypertension overloads the right ventricle, enlarges right heart chambers and ultimately causes right ventricular failure.

Left Ventricular Dysfunction

LV functions are usually normal in COPD patients.

Robotham et al[67] has found that LV dysfunction may be due to

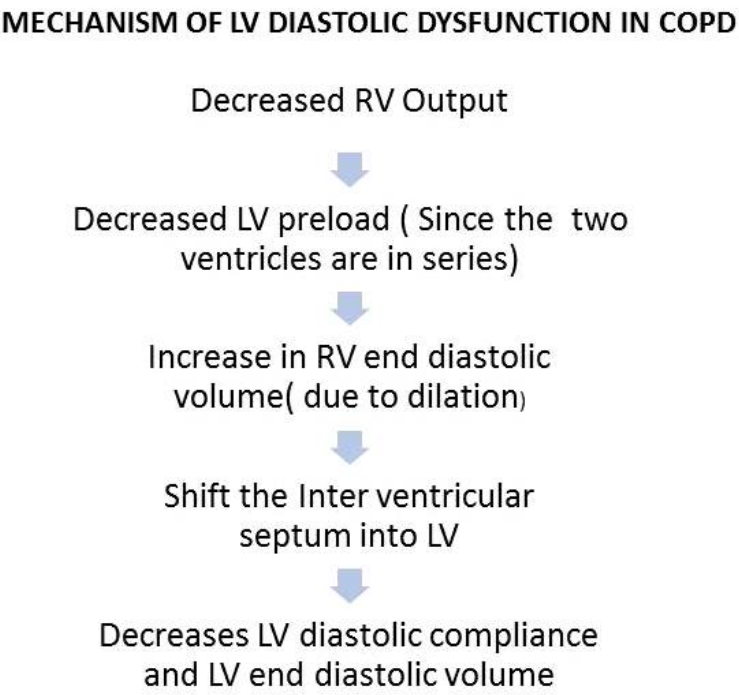
1. Hypoxia and acidosis

2. Reverse Bernheim phenomena or ventricular interdependence and

3. Large changes in intrathoracic pressure. (Due to hyperinflation, there is increase in negative pleural pressure which decreases LV stroke volume and increases afterload.)

In this study, LV diastolic dysfunction is found in 26 percent in the COPD group.

Louis EK et al[68] has demonstrated that probable cause of this LV DD is the increase RV volume or pressure which shifts the IVS into the LV and compromises the LV contraction, dimensions and compliance.



Schena et al[69] demonstrated that the right ventricular pressure correlates with left ventricular eccentricity indicies; the distorted ventricular geometry results in its abnormal filling pattern.

Butler et al[70,71] in his article, 'Heart in good Hands' said that "Hyperinflation and distension of lung increases the stiffness of parietal pleura and walls of cardiac fossa which adds load to both ventricles and contributes to right and left ventricular diastolic dysfunction in COPD patients".

In this study LV diastolic dysfunction(LVDD) is demonstrated in ECHO by E/A ratio and Iso volumetric relaxation time (IVRT). LV diastolic dysfunction is predominantly found in patients with elevated PAP.

Boussuger et al[72] also obtained similar results in 34 patients with moderate to severe COPD using combined analysis of mitral blood flow velocities and pulmonary venous blood flow velocities.

Tutar et al[73] described the left ventricular diastolic function impairment in COPD patients, and a similar correlation between right ventricular pressure, E/A ratio and IVRT were made out.

Mustapha et al[74] described the relation between right ventricular pressure and left ventricular diastolic dysfunction in a large group of cor pulmonale patients of different etiology (including COPD patients).

But Funk et al[75] showed that LVDD is found not only in patients with increased PAP, but also in normal PAP. It increases with RV afterload.

The elevated LV end diastolic pressure may be transmitted to the pulmonary system passively and causes exacerbation of dyspnea.

LV systolic dysfunction is not impaired in both COPD and control group in this study. Similar results were obtained by Schena et al[69] and Vonk-Noordergrat et al[76].

This shows that unless there is a primary pathology such as ischemic heart disease, hypertension etc. affecting LV systolic function, it is not compromised in COPD patients.

CONCLUSION

COPD can be considered as a preventable disease taking into account the percentage affected among smoking population in India.

The aim of this research being left ventricular involvement in COPD, the data collected from the study conducted that left ventricular diastolic function is compromised in COPD patients. Left ventricular systolic function remains unaltered even in advanced stages.

Suspicion and early diagnosis of left ventricular dysfunction in COPD is always required, ruling it out by proper evaluation is essential in reducing the morbidity.

Moreover, plethoric research must be conducted on extra pulmonary manifestations of COPD for wholesome understanding and management of the disease.

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PROFORMA

1. DETAILS

Name: Case No.:

Age: I.P. No.:

Sex: Unit:

Occupation : D.O.A:

Address: D.O.D:

Socio-economic status : Poor / Middle class/ Rich

Education : Illiterate / Literate

Locality : Rural / Urban

FINAL DIAGNOSIS

2. INFORMED CONSENT

I _____ unreservedly and in my full sense give

my full consent to take part in the study of lipoprotein abnormalities in chronic

renal failure. The risks and benefits have been explained to me in my

vernacularlanguage.

Date:

Signature of Patient / Attender

3. PRESENTING COMPLAINTS

1. Breathlessness Yes / No
2. Cough with expectoration Yes / No
3. Chest pain Yes / No
4. Palpitation Yes / No
5. Haemoptysis Yes / No
6. Fever Yes / No
7. Loss of weight Yes / No
8. Loss of appetite Yes / No
9. Puffiness of face Yes / No
10. Swelling of legs Yes / No
11. Decreased urine output Yes / No

4. HISTORY OF PRESENTING COMPLAINTS:

1. Breathlessness Yes / No
   - Onset: Sudden / Insidious
   - Duration: Hours / Days / Weeks/ Months / Year
   - Present at Unaccustomed work / Accustomed work/ Routine activity / At rest
   - History of PND Present / Absent
   - History of Orthopnea Present / Absent
   - History of fatigue Present/ Absent
2. Cough Yes / No
   - Duration: Days / Months / Years
   - Onset: Sudden / Gradual
   - Diurnal variation Present / Absent
   - Postural Variation Present / Absent

- Associated features — Wheezing / breathlessness

3. Expectoration — Present / Absent
4. Cough — Yes / No
   - Amount: — Mild / Moderate / Copious
   - Colour: — White / Yellow / Greenish / Rusty
   - Smell: — Foul Smelling / non foul smelling
5. Chest Pain — Yes / No
   - Onset: — Sudden / Gradual
   - Duration: — Hours / Days / Weeks / Months/ Year
   - Site: — Precardial / Lateral
   - Type of pain: — Squeezing / Pricking / Dull aching / heavy / burning / Catching compressing
   - Severity: — Mild / Moderate / Severe
   - Radiation: — Left arm / Neck / Abdomen / Back
   - Precipitated by — None / Exertion / Heavy meal
   - Relieved by — Rest / Drugs / None
6. Palpitation — Yes / no
   - Onset: — Sudden / Gradual
   - Duration: — Minutes / Hours / days
   - Rhythm: — Regular / Irregular
7. Heamoptysis — Present / Absent
   - Amount: — Mild / Moderate / Massive
8. Fever — Yes / No
   - Duration: — Days / Weeks / Months
   - Continuous / Intermittent
   - High grade / Low grade

- Associated features chills / rigors / evening rise of temperature

9. Loss of weight Yes / No
10. Loss of appetite Yes / No
11. Puffiness of face Yes / No
    - Duration: Days / Weeks / Months
    - Diurnal variation: Present / Absent
12. Swelling of legs
    - Duration: Days / Weeks / Months
    - Postural variation: Present / Absent
13. Urine out put
    - Amount: Polyuria Oliguria / Anuria
    - Duration: Days / Weeks / Months

5. PAST HISTORY

1. History of Tuberculosis Yes / No
    - Was Under CAT I / II / III
    - Treatment Completed / Defaulter
2. History of Asthma Yes / No
3. History of thoracic surgeries Yes / No
4. History of hypertension Yes / No
5. History of Diabetes Mellitus Yes / No
6. History of Heart Disease Yes / No
7. History of previous similar episodes Yes / No
    a. Number of acute exacerbations
    b. Use of inhalers Yes / No
    c. Use of bronchodilators Yes / No
    d. Use of Steroids Yes / No

6. FAMILY HISTORY

1. Contact history with Tb Yes / No
2. Hypertension Yes / No
3. IHD Yes / No
4. Diabetes Mellitus Yes / No

7. PERSONAL HISTORY

1. Diet Vegetarian/Non vegetarian/ Mixed
2. Smoking Yes / No
    - No. of Ciggarettes / Beedies per day
    - Duration
3. Alcoholic Yes / No
4. Sleep Normal / Disturbed
5. Menstrual history
    - Age of Menarche
    - Cycles
    - Menopause Attained Yes / No

8. GENERAL PHYSICAL EXAMINATION

1. Build Poorly built / Moderately built / Well built
2. Nourishment Poor / Good
3. Height (Cm)
4. Weight (Kg)
5. Body Mass Index (BMI)
6. Pallor Yes / No
7. Polycythemia Yes / No
8. Icterus Yes / No

9. Cyanosis Yes / No
10. Clubbing Yes / No
11. Pedal Edema Yes / No
    i. Unilateral / Bilateral
    ii. Pitting / Non pitting
12. JVP Elevated / Not elevated
13. Pursed lip breathing Present / Absent

9. VITAL SIGNS

1. Pulse
    - Rate
    - Rhythm Regular / Irregular
    - Volume Normal / *Low* / High
    - Character
    - Condition of vessel wall
    - Peripheral pulses Felt / Not felt
    - Radio femoral delay Yes / No
2. Blood Pressure
3. Respiratory rate
4. Temperature

10. SYSTEMIC EXAMINATION

Respiratory system:

- Upper Respiratory Tract
    - Nose
    - Sinuses
    - Pharynx

- Lower Respiratory Tract
- Inspection
  - Trachea — Right / Left / Central
  - Apical Impulse — Normal / Shifted
  - Movement of Chest Wall — Normal/ Increased/ Decreased
  - Skin over chest wall — Any sinuses / Discharge / Normal
  - Accessory muscles of respiration — Acting / Not acting
  - Dilated veins / Visible pulsation — Yes / No
  - Intercostal retraction — Yes / No
  - Spinal deformities — Yes / No
- Palpation
  - Trachea — Central / Right / Left
  - Apical Impulse — Normal / Shifted
  - Respiratory Movements — Normal / Decreased / Absent
  - Measurements

Expansion	Inspiration	Expiration
Right Hemithorax		
Left Hemithorax		

- Localised Swelling — Yes / No
- Intercostal tenderness — Yes /No
- Tactile Fremitus — Normal / Increased / Decreased
- Percussion
  - Lung fields

    Notes — Resonant / Impaired / Dull

    Specific Areas
  - Liver dullness in _____ space in Rt. MCL
  - Traubes space obliterated — Yes / No

- Auscultation
    - Breath Sounds

      Type - Vesicular / Bronchial / Bronchovesicular

      Intensity - Normal / Decreased / Increased
    - Added Sounds –

      Ronchi

      Crepitations - Fine / Coarse

      Pleural rub - Yes / No

      Whispering pectoriloquy - Yes / No

      Bronchophony - Yes / No

      Succussion splash - Present / Absent

CARDIO VASCULAR SYSTEM

- Inspection
    - Shape of Precardium
    - Apical Impulse Location
    - Pulsation present in
        - Aortic Area
        - Pulmonary Area
        - Parasternal Area
        - Epigastrium
        - Carotids
        - Back
- Palpation
    - Mitral area
        - Apex beat
            - Site and character

        - Palpable Heart sounds
        - Thrills
    - Tricuspid area
        - Left parasternal heave
        - Thrills
    - Aortic area
        - Palpable heart sounds
        - Thrills
    - Pulmonary area
        - Palpable Heart Sounds
        - Thrills
    - Carotid Thrills
    - Epigastric pulsation
- Auscultation

Areas	SI S2 S3 S4	Murmurs	Added sounds
Mitral			
Tricuspid			
Aortic			
Pulmonary			

    - Pericardial Rub Yes / No

ABDOMEN

- Inspection:
    - Shape - Normal / Distended / Scaphoid
    - Engorged veins - Present / Absent
    - Visible mass - Present / Absent
    - Visible peristalsis - Present / Absent

- Palpation:
    - Local rise of temperature - Present / Absent
    - Tenderness - Present / Absent
    - Palpable mass - Present / Absent
    - Organomegaly -
    - Fluid thrill - Present / Absent
- Percussion:
    - Liver dullness - Normal / Abnormal
    - Splenic dullness - Normal / Abnormal
    - Shifting dullness - Present / Absent
- Auscultation:
    - Bowel sounds - Normal / Decreased / Absent
    - Venous hum / Arterial bruit - Absent / Present
    - Splenic rub / Hepatic rub - Present / Absent

CENTRAL NERVOUS SYSTEM:

- Higher Mental Functions:
    - Level of consciousness
    - Appearance and behaviour
    - Emotional state
    - Orientation to time / Place / Person
    - Memory - Immediate / Short term / Remote
    - Speech
    - Handedness
- Cranial Nerves:

- Motor Functions:

  Right Left

  - Tone

    Upper Limb

    Lower limb
  - Power

    Upper Limb

    Lower Limb
  - Reflexes
  - Superficial

    Abdominal

    Cremasteric

    Plantar
  - Deep Tendon Reflexes

    Biceps

    Triceps

    Supinater

    Knee

    Ankle
- Sensory Functions:
  - Superficial - Pain / Touch / Temperature
  - Deep - Vibration / Position / Joint sense
  - Cortical sensation
- Cerebellar signs- Present / Absent
- Autonomic signs- Present / Absent
- Signs of meningeal irritation - Present / Absent
- Gait:

- Peripheral Nerves
- ANY OTHER FINDING

11. INVESTIGATIONS:

- Blood:
    - Hb%
    - TC
    - DC
    - ESR
    - Blood Urea
    - Serum Creatinine
    - Blood sugar
- Urine:
    - Albumin
    - Sugar
    - Microscopy
- Sputum AFB
- HIV 1 / 2
- Spirometry
    - FEV1
    - FVC
    - FEV1/FVC
- CXR- PA view
- ECG
- ECHOCARDIOGRAPHY

LIST OF ABBREVATIONS

COPD	-	Chronic Obstructive Pulmonary Disease
GOLD	-	The Global Initiative For Chronic Obstructive Lung Disease
WHO	-	World Health Organisation
TNFα	-	Tumour Necrosis Factor α
TGF-β		Transforming Growth Factor β
MMP	-	Matrix Metalo Proteinases
LTB4	-	Leukotrine B4
IL-6, 8, 10	-	Inter Leukines - 6, 8, 10
RV, LV	-	Right Ventricle, Left Ventricle
IC	-	Inspiratory Capacity
TLC	-	Total Lung Capacity
FEV1	-	Forced Expiratory Volume In 1 Second
FVC	-	Forced Vital Capacity

BODE INDEX	-	Body Mass Index, Airflow Obstruction, Dyspnea, Exercise Capacity
ACE	-	Angiotensin Converting Enzyme
PPAR	-	Peroxisome Proliferative Activated Receptor
PAP	-	Pulmonary Arterial Pressure
RVSP	-	Right Ventricular Systolic Pressure
TVPG	-	Tricuspid Pressure Gradient
RAP	-	Right Atrial Pressure
EF	-	Ejection Fraction
%FS	-	% Fractional Shortening

MASTER CHART OF STUDY GROUP

S NO	NAME	Age	Sex	FEV1	FVC	FEV1/FVC	GOLD COPD STAGING	CORPUL MONALE	ELEVATED PAP	LVDD	LVSD
1	CHELLAKANNU	45	F	0.7	1.8	44	SEVERE	A	P	P	A
2	RAJENDHIRAN	50	M	1.2	2.1	57	MILD	A	A	A	A
3	PAAPA	60	F	1	1.8	44	MILD	A	P	A	A
4	NAGARAJAN	40	M	1.3	2	68	MILD	A	A	A	A
5	SATHYAMOORTHI	42	M	0.8	1.7	77	SEVERE	A	A	A	A
6	AYYAKANNU	50	M	0.9	1.9	47	MODERATE	P	P	P	A
7	KANDHAYEE	61	F	1.2	2.1	57	MODERATE	A	A	A	A
8	AMUTHA	50	F	1	1.8	55	MILD	A	A	A	A
9	KRISHNASAMY	39	M	1	1.9	53	MODERATE	A	P	P	A
10	ANGAMMAL	50	F	1.1	2.4	48	MILD	A	A	A	A
11	LAKSHMANAN	36	M	1.2	2.1	57	MODERATE	A	A	A	A
12	KALIANNAN	36	M	0.9	1.8	50	MODERATE	A	A	A	A
13	MADHAMMAL	41	F	1.9	2.3	61	MILD	A	P	P	A
14	THANGAVEL	49	M	1.1	2.2	50	MODERATE	A	P	A	A
15	DHANAKODI	39	F	1	2.1	47	MODERATE	A	P	A	A
16	RAJENDIRAN	49	M	1.2	2.1	57	SEVERE	P	P	P	A
17	KANNAN	44	M	0.5	1.4	36	VERY SEVERE	P	P	P	A
18	BAKKIAYAM	46	F	2.1	3.1	67	MILD	A	A	A	A
19	GOVINDAMMAL	50	F	0.8	1.4	57	MILD	A	A	A	A
20	ANBARASU	37	M	0.7	1.8	44	MILD	A	A	A	A
21	SHANTHA	54	F	1.3	2.3	56	MILD	A	P	A	A
22	VENKATESAN	41	M	1	1.8	55	MODERATE	A	A	A	A
23	KAMALAM	51	F	1.2	2.1	57	MODERATE	A	A	A	A
24	ARUMUGAM	60	M	1	2	50	MODERATE	A	P	A	A

S NO	NAME	Age	Sex	FEV1	FVC	FEV1/FVC	GOLD COPD STAGING	CORPUL MONALE	ELEVATED PAP	LVDD	LVSD
25	MADAMMAL	60	F	0.9	1.8	50	SEVERE	A	A	A	A
26	KANDASAMY	42	M	1	1.8	56	MODERATE	A	A	A	A
27	ANTONY RAJ	36	M	1	1.8	55	MODERATE	A	A	A	A
28	KAVIARASI	45	F	1	2.2	49	MODERATE	A	A	A	A
29	VEDIYAMMAL	40	F	1	1.7	59	MODERATE	A	P	P	A
30	KAVERIYAMMAL	42	F	0.9	1.8	50	MODERATE	A	A	A	A
31	MUTHARASU	46	M	0.9	2.1	46	MODERATE	A	A	A	A
32	ARASAPPAN	36	M	0.9	2.1	46	MODERATE	A	A	A	A
33	SHANKAR	52	M	0.7	1.7	41	SEVERE	P	P	P	A
34	GANESAN	50	M	1	1.8	56	MODERATE	A	A	A	A
35	RAJESWARI	46	F	0.8	1.9	50	SEVERE	A	P	A	A
36	KULANDAIVEL	56	M	1.1	2.4	48	MODERATE	A	A	A	A
37	JAGANADHAN	57	M	1	1.9	52	MODERATE	A	P	P	A
38	ARULMANI	49	F	0.9	1.8	50	MILD	A	A	A	A
39	SUNDARAJAN	44	M	0.6	1.6	37	VERY SEVERE	A	P	P	A
40	AROKKIYASAMI	59	M	0.7	1.7	38	MILD	A	A	A	A
41	ANBALAGAN	74	M	1	1.8	55	MODERATE	A	A	A	A
42	IRUSAGOWNDER	75	M	0.7	1.7	41	MILD	A	A	A	A
43	MEENAKSHI	51	F	1	1.9	52	MODERATE	P	P	P	A
44	RANGANAYAGI	60	F	0.7	1.7	41	VERY SEVERE	P	P	P	A
45	VALLI	52	F	1	1.9	52	MODERATE	A	A	A	A
46	BASHEER	42	M	1	1.8	55	MODERATE	A	A	A	A
47	PALANIYAMMAL	47	F	0.9	1.8	50	MODERATE	A	A	A	A
48	BALAJI	45	M	0.6	1.5	40	VERY SEVERE	P	P	P	A
49	KANNAPPAN	56	M	0.7	1.8	44	MILD	A	A	A	A
50	JAYAMARY	60	F	0.9	1.9	48	MODERATE	A	A	A	A